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XVIII.

HEMATOLOGIC INVESTIGATIONS ON SOME IN-  
FECTIONS IN THE THROAT AND IN CHIL-  
DREN WITH ADENOID GROWTHS.

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COPENHAGEN.

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## PREFACE.

While the investigations of the blood picture in scarlatinous angina were commenced as early as in 1918, the more essential part of this work was begun in 1920. It was my idea, through a more extensive material, to follow the plan which was devised by the senior physician at the Blegdam Hospital, Professor V. Bie, M. D.—a plan that was begun by Skat Baastrup, M. D.—viz., to see whether the difference in the blood picture of scarlatinous angina and ordinary angina is also present in mild cases of scarlatinous angina where the diagnosis cannot be made until after a shorter or longer time of observation, to the end that the diagnosis of scarlatinous angina may possibly be supported or rejected by hematologic examination.

A number of patients admitted to hospital under observation for scarlatinous angina were found to be suffering from other infections than scarlatina, and it was therefore thought that, even if it would prove an extensive task, it would be possible, by means of a large number of investigations, to assemble clinical cases which would yield a hematologic picture of a number of the more common infections in the throat and air passages.

I found both material and working conditions I wanted at the Blegdam Hospital and at the otolaryngologic department of the University Hospital, and I beg Professor V. Bie, M. D., and Professor E. Schmiegelow, M. D., to accept my cordial thanks for having requested me to undertake this work. Furthermore, I wish to thank Mr. S. H. Mygind, M. D., senior physician at the Kommunehospital, for permitting me to undertake a minor part of the examinations at the otolaryngologic department of

this hospital. I am especially indebted to the astronomer, Mr. S. Fjeldtofte, who with great kindness has supervised my calculations.

I have determined the blood picture in a number of normal adults and normal children; this, however, has been treated more elaborately in earlier works. By these examinations I intended to establish a basis, and as these small series of examinations in all essentials were in harmony with the results obtained by previous authors, I have limited myself to a minor number of examinations. As a special chapter, the determination of the blood picture in children with adenoid growths, as compared with that of normal children, is included.

#### HISTORICAL REVIEW.

I. (a) Development of the Counting Methods, (b) Static Leucocytic Reactions. II. Digestion Leucocytosis. III. Hemoclastic Crisis.

The first endeavors to count leucocytes were made as a secondary undertaking while counting red corpuscles, the leucocytes being counted together with the red cells, on account of the great uncertainty in discerning which were leucocytes and which erythrocytes and with an inexactitude that made these counts rather insignificant. Wagner (according to Sørensen) was the first (1838) to try to determine the relation between the leucocytes and the red corpuscles in a drop of blood spread under a cover glass. In 1854, Moleschott published a work on the relative numbers of red and white blood corpuscles, and next year de Pury examined the relation of the leucocytes in cases of intermittent fever, finding an increase of the number of leucocytes during the febrile stages. The erythrocytes and leucocytes were counted in a drop of blood of no fixed size. On account of the exceedingly trying work of counting such a great number of red cells, in view of the small number of leucocytes, threads or lines were placed in the ocular. This was the origin of the division of the fields of vision for counting blood cells. In 1853, Welcker thought of destroying the red corpuscles by diluting 25 times with distilled water. In addition to overcoming the difficulty in finding all the leucocytes, which, in the method previously used, were often hidden by the numerous red cells, he was the first to de-

vise a mode of dilution by which he could count up to one thousand leucocytes, and in contradistinction to earlier investigators, he determined the number in one cubic millimeter of blood. In 1873, Malassez published his mode of counting, which became the leading and very useful method, but only for a short time, until Thoma, in 1878, brought forward his counting chamber, constructed by Zeiss. Malassez's method was based on the "mélangeur," invented by Potain in 1867, for dilution of blood and uniform distribution of the blood cells. The principle of this "mélangeur" was retained, until another stride ahead, as to accuracy and handiness, was made with Ellermann and Erlandsen's separated pipettes and mixing glasses. Malassez constructed a flat cut capillary tube, cemented to a slide. The blood mixture from Potain's mélangeur (the blood is diluted with a gum solution) is drawn into this capillary tube, by capillary attraction or by suction, from the opposite end of the capillary tube. The division lines were placed in the ocular, and the lumen of the capillary tube was measured by means of filling and weighing mercury. With this method as a basis, a valuable work was published in 1876 by the Dane S. T. Sørensen, the special aim of which was to count red corpuscles; as to leucocytes, S. T. Sørensen, who himself mentions the inaccuracy of these counts, found, however, values which are in accordance with later determinations; in cases of typhoid fever, a decrease was found, whereas the number of leucocytes in pneumonia, in puerperal fever, rheumatism, etc., in proportion to the red cells, had increased to 4, 6, 7 and even, in a few cases, to 10 times the usual amount. In normal persons the leucocyte count showed values of 6,600 to 7,300 per cm. With the acetic acid method, devised by Thoma in 1882 for destruction of red corpuscles, in connection with his counting chamber, which appeared in 1878, the counting of leucocytes slipped into the groove in which it remained for the following 20 to 25 years, no special new methods being invented during this period; investigators have busied themselves in devising new forms of counting chambers. These endeavors were not futile, as the small, ruled counting in the Thoma-Zeiss chamber gave rise to great errors in counting. Of counting chambers appearing during the following years I want to mention those constructed by Zappert,

by Elzholz and by Bürker-Türk, the latter being a double chamber, into which the well shaken blood mixture is drawn by capillary attraction, a principle which has been adopted latterly for counting leucocytes in other counting chambers; the cover glass is first pressed down on the empty chamber, a small area of the counting chamber, however, being left uncovered; in this area is placed a drop of the blood mixture, which is then sucked in under the cover glass. Mention must also be made of Breuer's chamber, which has a Thoma-Zeiss field in the middle, surrounded by eight similar fields, each of which is divided only in four parts by three cross lines. The purpose of Breuer's chamber is to let the area of the counting field increase in size and thus to some degree adjust the error caused by the unequal distribution of the leucocytes in the small area.

The Fuchs-Rosenthal counting chamber has the same advantage. It was originally constructed to count leucocytes in the cerebrospinal fluid, where, in most cases, only a comparatively small number of leucocytes will be found. For this reason the chamber is also constructed of double the height,  $2/10$  mm., as compared with  $1/10$  mm. in the above mentioned chambers.

Kjer-Petersen, whose merits as to the introduction of the mean error determination in leucocyte counts will be mentioned later on, realized the weak point of the small counting areas. With a low magnification (70 times) he therefore counts in fields, the division of which is found in the ocular; thus in this respect he applies the same principle as that used by Welcker and Malassez.

Before describing the period during which the study of the physiologic variations in the number of leucocytes was the main object in hematologic investigations—a period initiated with Kjer-Petersen's assertion of nonhomogeneity in women's blood—a number of results of leucocyte counts in normal individuals, in which the different counting chambers have been used, will be mentioned; already at an early stage these examinations brought the problems of the diurnal variations to the forefront of the discussion.

Malassez found values ranging between 3,700 and 7,700 leucocytes per cm., while some French authors found highly varying figures between 2,000 and 10,000. Sörensen, as men-



tioned above, found figures fluctuating between 6,600 and 7,300. In 1882 Thoma and Havem found values varying from 5,000 and 6,500 to 8,300, in normal individuals. Reinecke and Reinert (1889 and 1891) examined normal individuals at different times of the day. While the former found nearly uniform values throughout the day (7,107, 7,482, 7,464), calculated from the average values, the latter found the average values to be highest at 4 p. m. (8,200, compared with 5,100 at 6 a. m.), after which time there was a slight fall to 7,200 to 6,000, at 8 and 10 p. m., respectively. v. Limbeck (1890), like Reinert, found an increase in the number of leucocytes in the course of the day dependent upon a digestive leucocytosis. The average value found by him in men and women was 8,500. It should be stated that Moleschott, before Virchow, mentioned the possibility of a digestive leucocytosis. Virchow suggests that the mesenteric glands, after the meal, are placed in a state of irritation by the chyme supplied by the intestines, and that at the same time a physiologic increase of the number of leucocytes in the blood takes place.

Tornow (1895) found variations in the number of leucocytes subsequent to psychic influences, and also an increase in soldiers after strenuous marches.

Bruhn-Fähræus (1897), who also saw increases during daytime, found that the increase was independent of the meals. He made some of his subjects take their meals at different times of the day, and he made others fast, and yet he found uniform curves for the different groups. On an average he found 6,000 leucocytes in men and 7,050 in women, two to three hours after breakfast. Japha (1900) also found a hyperleucocytosis in the middle of the day, even during a period of fasting.

Although Bruhn-Fähræus had computed his counting error, Kjer-Petersen, as mentioned above, was the first (1905) to determine his mean error, and also estimated his figures according to the laws of observation and thus fulfilled the requirement in counting leucocytes, that the examiner should know which of the values found fall within and which fall without the limit of error—in other words, which values indicate a true increase or decrease, and which differences in value do not really signify anything. Kjer-Petersen's work in

several respects shows certain defects, according to modern opinions, e. g., the application of a low magnification (70 times), which seems to prevent the possibility of determining with any certainty what are leucocytes and what are casual impurities. Furthermore, later investigators have rejected the claim of the nonhomogeneity in women's blood. It has in its highly limited material in the main section (five men) both an advantage and a drawback—an advantage because these subjects have been examined daily through weeks and months, and a disadvantage because, as stated by Oluf Thomsen, it is not possible by producing over and over again a small wound in a certain limited area (the earlap) to guard oneself against an inflammatory reaction in the tissue with an accumulation of leucocytes in the surrounding capillæ. Oluf Thomsen is of opinion that this last mentioned phenomenon may explain Kjer-Petersen's nonhomogeneity, while Ellermann and Erlandsen find the explanation in psychic influences and static reactions. Kjer-Petersen finds that the diurnal increase in the number of leucocytes runs parallel with the increases in the pulse and temperature curves. He finds that the diurnal increase begins before the first meal, and that muscular work increases the number of leucocytes. Immediately after awakening, the number of leucocytes in adult men was found to lie between 4,000 and 5,000. Moreover, he finds that even slight degrees of catarrhal states (cold) bring about an increase in the number of leucocytes.

In a very extensive work, A. von Bonsdorff found, in 30 healthy men and 30 healthy women, between the ages of 18 and 25 years, that before taking any meal the number of leucocytes early in the morning lay between 4,500 and 7,500. He also found that even a relatively slight pathologic state, such as an acute catarrh of the mucous membranes, may cause an increase of leucocytes. Moreover, he found variations in the number of leucocytes in the same persons from day to day, which, it is true, in most cases fell within the limit of error, but in many cases fell without it, as an expression of a true variation.

In mentioning the above works no exhaustive account of the works written before 1905 treating upon the normal values and the variations of leucocytes under different conditions has

been given, but only a few main points from the available literature have been mentioned, the object of this treatise not being to determine the number of leucocytes in normal individuals and under certain physiologic conditions. For the same reason only the main works on static leucocytic reactions which have appeared after, and no doubt partly have been called forth by, Kjer-Petersen's assertion of nonhomogeneity will be mentioned in the following.

#### STATIC LEUCOCYTIC REACTIONS.

Running parallel with the well known increase of the pulse rate found at sudden changes from the recumbent to the vertical position, Hasselbalch and Heyerdahl found a hypoleucocytosis, and, vice versa, by a change from the standing to the recumbent posture—corresponding to the decrease in the pulse rate—an increase in the number of leucocytes in the dermal vessels, sometimes amounting to upwards of 100 per cent, but lasting only up to a few minutes. They found that polynuclears and mononuclears to the same degree took part in the increase. In patients with heart disease, the reverse was sometimes found, both as to the number of leucocytes and the pulse rate. Moreover, they found that a local hyperemia in the dermal capillaries does not influence the number of leucocytes in the said area to any essential degree. After a short exhausting run of about 10 minutes, a hyperleucocytosis was demonstrated, being somewhat more pronounced in the capillary blood than in the corresponding venous blood. In five normal persons who had all been previously examined for static leucocytic reactions, the arterial pressure in the arm was found to be highest in the recumbent position, lowest in the standing posture (difference 40 to 60 mm.). The leucocytic reactions occurred within the same short period in which the difference in pressure was found. The authors maintain that the difference between the systolic and diastolic blood pressure—a difference which is diminished by a sudden change from the recumbent to the erect posture and is augmented by the opposite movement—is the cause of the static leucocytic reactions. In 23 experiments, Ellermann and Erlandsen observed the static leucocytic reactions shown by Hasselbalch and Heyerdahl, who did not find them constant, and they show that the static leuco-

cytic reaction is maximum between 15 and 45 seconds, and often has passed after 45 seconds; here, they think, is the explanation that this reaction was not constantly found in Hasselbalch and Heyerdahl's experiments.

The passing increase in the number of leucocytes occasioned by muscular work, and shown by earlier authors (Schultz, Grawitz, Hasselbalch and Heyerdahl), was studied by Ellermann and Erlandsen, who found the increase constant also at a repetition of the muscular work. On the basis of their investigations Ellermann and Erlandsen are of opinion that it is here a question of the same phenomenon as in the case of static leucocytic reactions; a changed activity of the heart causes the leucocytes to move from a peripheral depot to the peripheral vessels.

Ellermann and Erlandsen give the same explanation of the hyperleucocytosis found as a result of psychic irritation, and this phenomenon—the passing hyperleucocytosis in consequence of a psychic irritation, which may involve a source of error in clinical examinations in case the first drop is used for counting—has, according to the experiments made by Ellermann and Erlandsen, caused the introduction of the technical rules, without which countings of leucocytes will give inaccurate results, viz., to make the cut in the earlap somewhat before taking the sample for counting the leucocytes, and not to use the first drop. Ellermann and Erlandsen once having drawn attention to this fact, it will be possible for everybody concerned with leucocyte counts to persuade himself that the first drop very often contains more leucocytes than the next one.

Another form of physiologic leucocytosis, which it is necessary to eliminate at the clinical examinations, is that due to thermic influences. It is true that the experiments made by Winternitz, Knöpfelmacher and others, where an increase of leucocytes is found in the capillary blood after baths by which the body temperature is diminished, cannot be directly compared with the influence of the cold to which the patients are exposed, e. g., when walking to the hospital in winter, such as has been the case with part of the patients in our material. Yet it can hardly be doubted that the contraction of the dermal vessels which takes place in persons who are exposed to cold weather must be able to call forth the same phenomenon—

although presumably to a less extent than when the dermal capillaries contract in the above mentioned cooling experiments, viz., an accumulation of the leucocytes in the peripheral capillaries, whether this phenomenon is due to a different distribution of the leucocytes in the different vascular areas or to alteration in the concentration of the blood.

In a number of experiments, in which the first sample of blood was taken immediately after the patient came from the open air into the hospital on a cold winter day, values for leucocytes were found which were nearly 30 per cent above those found when the patient had been sitting for a quarter of an hour in room temperature. It is possible, here, that two different factors have made themselves evident, viz., partly a thermic leucocytosis, partly a myogenous leucocytosis in consequence of the muscular work which took place during the patient's walk to the hospital. On the basis of these experiments, a permanent rule has been introduced for outpatients, viz., to let the patients remain quiet for not less than a quarter of an hour in room temperature before the sample is taken. Experiments made to find out whether a further waiting time might bring about any alteration in the number of leucocytes constantly showed that this was not the case.

Before accounting for the quite recent works on the variations in the number of leucocytes in certain physiologic conditions, I want to mention a Danish work, in which the theoretic reasons of the static leucocytic reactions have been more scientifically dealt with. It is Gustav Jörgensen's experimental investigations on transitory variations in the number of leucocytes following injections with strophanthin (1915). Similar experiments had already been made by Ellermann and Erlandsen by using 1/10 mg. of strophanthin crystal per kilogram on urethanized rabbits. By means of a cardiometric registration of the pulsation of the heart, Gustav Jörgensen shows a fully parallel course of increase of the contraction of the heart—and the increase of the blood pressure connected therewith—and a transitory hyperleucocytosis which was not associated with any change in the blood picture as regards polynuclear and mononuclear leucocytes, nor with any change in the number of red cells, which showed that the leucocytosis could not be

due to alterations in the concentration of the blood. Apart from the results found, Gustav Jörgensen's work, which was continued in 1917, is of very great interest on account of his mean error determination. He works with the counting method given by Ellermann and Erlandsen, with a mean error which, when counting 100 fields with 115 leucocytes, is 6.3 per cent of the average. While Gustav Jörgensen, just as Ellerman and Erlandsen, uses for diluting fluid equal portions of a 1/10 per cent solution of normal hydrochloric acid and a physiologic solution of sodium chlorid, to which is added formalin (1 portion of formalin to 9 portions of the above mentioned fluid), and also uses the method with separated pipettes and mixing glasses devised by Ellermann and Erlandsen, he has preferred the Leishman stain of the smear preparation which, in Ellermann and Erlandsen's method, is stained with 1 per cent of an aqueous solution of methylene blue, to which is added a soda dilution in order to obtain an alkaline reaction during the staining, as the hydrochloric acid of the diluting fluid is not an ideal staining fluid, especially on account of precipitates.

No doubt this method is the most exact counting method hitherto applied, and it is also very useful for experimental investigations in which there is only a question of differentiating between polynuclears and mononuclears in the dry preparation. In the chapter on the technics applied, account will be given of the reasons why counting chamber and stained films have been preferred in the present work, as I have adopted, in the technical mode of procedure, the principle of separated pipettes and mixing glasses. In regard to the spreading of the fluid in the counting chamber, I have followed the technical devices on which, according to my personal experiences, I have reason to suppose that the accuracy of Ellermann and Erlandsen's method rests.

The development of certain questions to which regard must be paid in the daily clinical work still remains to be accounted for, viz., alterations in the total numbers and the qualitative relations of the leucocytes, partly under certain physiologic conditions, which have been mentioned only incidentally in the preceding pages, partly under certain pathologic conditions,

i. e., alterations in the blood picture (a) during digestion, (b) under certain anaphylactic\* conditions.

While mentioning the development of the counting methods, it has already been necessary to touch upon the first assumptions as to the existence of a digestion leucocytosis, as the very experiments to find improved methods went hand in hand with the investigations of certain physiologic variations in the number of leucocytes.

I shall just briefly mention that Moleschott, Virchow, v. Limbeck and Reinert, also Hirt, Sørensen, de Pury, Rieder, van Noorden, Zappert, Hoffmann, Labbé-Besancon, Sirensky, Lerenski and Pappenheim found an increase of the number of leucocytes during the first hours after a meal, more especially after a meal rich in albumen. By experiments with animals, Pohl found alimentary leucocytosis in dogs after albuminous meals, while Goodall, Gulland and Paton in cats and dogs found such an increase both after fats and albumen, and, more especially, they found an increase of the lymphocytes, while several of the above mentioned authors, who made differential counts, found either a proportionate distribution in the increase of polynuclears and mononuclears or a preferential increase of the polynuclears (v. Limbeck, Sirensky, Pappenheim). Schwenkebecker and Siegel also found digestion leucocytosis in animals, while Klieneberger and Carl failed to find any alimentary leucocytosis in dogs and cats and a number of rodents.

A number of authors, for instance, Halla, Hayem, Malassez, Reinecke, Galambos, Rothacker, Bruhn-Fähræus, Ellermann and Erlandsen and Naegeli, did not find any alimentary leucocytosis in man. In Naegeli's textbook, "Die Blutkrankheiten" (1920), it is said that digestion leucocytosis is one of the most obscure problems within morphologic hematology, while Pappenheim in his textbook (1921), on the basis of the works extant, maintains that albuminous meals call forth a polynuclear leucocytosis.

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\*In the following the term "anaphylaxis"—pursuant to the literature—is used, while, according to more modern views, the term "allergia" ought to be used.



Arneth, who in a more extensive treatise (1920) accounts for an alimentary leucocytosis after albuminous meals with special increase of the polynuclear leucocytes, joins the adherents of a digestion leucocytosis. By means of the counting of nucleal fragments introduced by him—a method the significance of which is not admitted by Pappenheim—he finds no shifting within the five classes of polynuclears, especially no “turning to the left,” i. e., no increase of the youngest polynuclears; he infers from this fact that the addition of polynuclears to the circulation does not depend on a neoformation from the bone marrow, but is due to a supply from the depots of polynuclears present in the organism.

In a later work (1923), this question is again taken up in collaboration with Ostendorf; in five healthy men a maximum increase of 35 per cent is shown three hours after the ingestion of albuminous food, exclusively involving the polynuclears, without any change in Arneth's blood picture. The experiments were made by giving 250 gr. of meat on an empty stomach.

It seems to me that many of the experiments made point to a true digestion leucocytosis of polynuclear type, after liberal ingestion of albumen, in spite of the fact that the numerous works dealing with digestion leucocytosis do not, at any rate as far as the majority are concerned, account for the calculation of errors in the numerical and differential counts, no more than they pay any regard to static and psychic reactions—although it is probable that the experiments have been made under the proper conditions—and, in spite of the fact that, as especially shown by Ellermann and Erlandsen, a diurnal increase no doubt exists independent of the meals.

Among the investigations into the alimentary leucocytosis, the conditions found in infants take up an exceptional position. It must immediately be pointed out that a number of authors have come to very different results: Moro, e. g., in nine healthy suckling infants, found a leucopenia which was not compensated until after three to four hours. Wernstedt, in 27 breast-fed infants, found no regularity in the variations of the leucocytes during and after lactation. Salzberger found leucopenia in breast fed infants after preceding inanition; this was also found by Gundobin until three hours after the meal. Schiff

and Stransky also found a passing leucopenia both after use of woman's milk and after cow's milk.

Hutinel was the first who interpreted idiosyncrasy for cow's milk in infants as an anaphylactic phenomenon. This view was later on accepted by Richet, but not until Danysz showed that the albuminous substances absorbed through the intestinal canal were partly reabsorbed by the blood as albumoses and peptones without being resolved into aminoacids was the alimentary anaphylaxis correctly recognized. These questions gave rise to a number of investigations and views within pediatry, especially by Americans.

Diagnostics of alimentary anaphylaxis has had special support from investigations on the leucocytes. Preceding the theory of an alimentary anaphylaxis a number of investigations on alterations in the blood picture following a parenteral ingestion of albumen had been undertaken. As early as 1902 Richet produced an anaphylactic shock in dogs by injection with aktinokongestin, and during this shock he demonstrated a great and sudden decrease in the blood pressure. Widal, Abrami and Brissaud (1913) showed that the paroxysmal hemoglobinuria was accompanied by leucopenia with almost complete disappearance of the polynuclears, a phenomenon which three years before had been noted by Biedl and Kraus as being decisive of the anaphylactic state. The two authors point out that it is not a question of a true leucopenia, but of a different distribution in the different vascular areas with an accumulation in the pulmonary vessels of polynuclear leucocytes.

The paroxysmal hemoglobinuria stands as a paradigm of a clinically anaphylactic state, but is without interest where it is a question of an alimentary anaphylaxis, or, at any rate, where there is no distinct connection between nutrition and the attack. Another condition is found, which to a higher degree seems to be in relation to the diet, viz., the exudative lymphatic diathesis, with its periodic dermal eruptions (urticaria, strophulus) and acute irritations of the mucous membranes (pharyngitis, rhinopharyngitis, acute rhinitis, laryngitis, bronchitis, asthma). The acute exacerbations in this disease are often accompanied by lymphocytosis and eosinophilia. Among the different symptoms the asthmatic paroxysms are of special

interest, as, both in adults and in children, they are elicited by different substances which irritate the air passages (the dust of certain grasses, feathers, animal hairs, etc.); they may also in certain cases be a consequence of the food, and thus may be considered as called forth by an acute alimentary anaphylactic state. Even if, of late, there has been a tendency to ascribe too much importance to the alterations in the blood picture in the presence of an anaphylactic state, the blood pictures found in asthma and in a long series of acute dermal conditions belonging to the exudative lymphatic diathesis seem to corroborate the view that it is a question of an anaphylactic state.

Recent studies on the relation of the leucocytes to the absorption of food have especially centered round an alimentary leucocytic reaction (Widal's test, or "Leuco-Widal," as it has been called by Schilling), the physiologic basis of which should be the reaction of the organism against penetration of heterogeneous albumen into the blood; and it is supposed that morbidly altered liver parenchyma would permit albumen in the form of albumoses and peptones (from 200 to 300 gr. of milk ingested after an inanition of at least five hours), at any rate partly, to pass into the blood and there call forth the so-called hemoclastic crisis within a period of 20 to 50 minutes. This crisis is characterized by a great decrease in the number of leucocytes especially, due to the disappearance of the polynuclears from the peripheral circulation. The value of this test has not yet been established; it must be mentioned that Bauer found a positive reaction in patients suffering from a liver disease. Holzer, Schilling had the same results in patients suffering from an intestinal disease without any hepatic complications. Kisch, Philipp and Jungmann found the reaction, although not so constant, after the administration of animal and vegetable fat and sugar to patients suffering from liver diseases. It is of special interest that the reaction seems to be constant after salvarsan poisonings, in which cases it is known that an acute parenchymatous hepatitis exists. Schiff and Stransky, and also Glaser, find that infants have an alimentary leucopenia. The explanation should be that the hemoclastic crisis is a vague reflex and that children, from a physiologic point of view, are vagotonics. By the aid of adrenalin it

should be possible to change the alimentary leucopenia to a leucocytosis.

As a short summary of this chapter, which from practical reasons treats of the development of the counting methods and of the most important problems of physiologic leucocytosis side by side, it may be stated that the technic of the blood cell count has been developed so far that, when excluding the above mentioned physiologic variations and introducing the mean error determination we are now able to estimate what is a true variation in the number of the leucocytes. The importance of the earlier works, however, must not be underrated; for instance, investigators early realized the importance of making the counts in the morning before the patients had partaken of any meal or had performed any movements, and they also realized the fundamental ideas of the physiologic variations. Danish investigators were in the front rank among those who cleared up the problem of the static variations until the views now in force gained ground, and it was the same investigators who improved the counting methods. However, it still seems as if the many interesting publications, which of late years have appeared abroad, and of which several of the most important ones have been mentioned above, do not pay sufficient regard to the mean error determination. It is evident that if it is desired to be on absolutely safe ground all variations which fall within the range of four times the average error or, at any rate, within three times the average error, ought not, pursuant to the exponential law of errors, to be considered as true increases or decreases, inasmuch as they may be counting errors. The consequence is that a long series of observations should be rejected, as it is not known whether they indicate true alterations in the number of leucocytes. If 6,000 leucocytes are counted, allowing for an error of 7 per cent, it means that it is possible to get a deviation in both directions of 1,680 leucocytes (28 per cent of 6,000), so that the figure may be 4,320 just as well as 7,680. Thus, for instance, the majority of the works on alimentary leucocytosis would be without any value, as it is a question of increases of 30 to 40 per cent. In such scientific investigations, however, it is certainly reasonable to suppose that there is a true increase, even if it falls within the limit of error, in case the

increase proves to be constant in a long series of experiments, and if the values constantly point in the same direction, e. g., before a meal, 4,300, half an hour after the meal, 4,800, one hour after the meal, 5,200, etc., all the time bearing in mind that it is only a question of probability, it will not be possible to obtain more reliable results before we can work with a method which involves so small an average error that the values of the increase fall outside its limit.

## CHAPTER II.

### THE TECHNIC APPLIED.

- A. Determination of the total number.
- B. Differential count.

#### A. DETERMINATION OF THE TOTAL NUMBER.

In the preceding chapter the main lines of the development of the counting methods have been established, with the opinion that the greatest accuracy is attained in the method devised by Ellermann and Erlandsen. In the preparation stained in accordance with this method, leucocytes are easily discernible from mononuclears, but, although the protoplasm of the eosinophiles is seen as a bluish red shade, it is hardly possible with regard to these cells to obtain so reliable results as in the usual smear and staining methods (Leishman-Giemsa, May-Grünwald). Nor within the group of mononuclears is it possible reliably to discern lymphocytes from transitional forms and large mononuclears, a fact which perhaps is of minor importance, as opinions of late more and more seem to agree that these different cells belong together, both from a genetic and from a physiologic point of view. It is desired, in the present work, to differentiate only between four main groups in the hematologic investigations:

(1) Polynuclear neutrophiles, (2) polynuclear eosinophiles, (3) lymphocytes (comprising large and small lymphocytes), and, as the fourth group, (4) monocytes, comprising both transitional forms, as also great mononuclears. It will therefore prove expedient to adhere to the old principle and count in a stained film.

In the present work, the following mode of procedure is used:

(1) Ellermann-Erlandsen's principle in mixing glasses and separated pipettes.

(2) Counting chamber, in which the spreading of the fluid is made according to the same principles as those used by Ellermann and Erlandsen when spreading the fluid on the albumen glycerin film in the dry preparation.

(3) Stained film according to the technical principles used in recent years.

As to the first, the mode of procedure has been the same as that stated by Ellermann and Erlandsen. A calibrated pipette is used, a mark corresponding to 25 mm. of blood, taken from a freely bleeding incision in the earlap made a little before the sample is taken; the first drop is not used. The blood is blown out into a mixing tube of nearly the same size and form as a Wassermann tube, and in which is placed, together with a small glass ball, 475 mm. of a 3 per cent dilution of acetic acid, to which is added methyl violet. After making various tests it appeared that the stained leucocytes were most clearly visible in the counting chamber when a very thin dilution of methyl violet (1:5,000) was used. It is necessary to make sure that the acetic acid, which may be kept for months, does not contain precipitates of methyl violet or fungi (blastomycetes) which, to the untrained examiner resemble red corpuscles, and, if found in great numbers, may completely compromise the accuracy of the counting. I have always found the hemolysis to be complete. Moreover, a skilled observer will never mistake erythrocytes for leucocytes. The drop of blood is allowed to settle; the pipette is now washed with a little of the mixing fluid, which is then blown into the mixing glass just at the level of the fluid, by which blood and diluting fluid become mixed. The last little drop from the pipette is placed on the inner side of the glass, which is closed with a well adapted gum plug and shaken to hemolysis.

(2) For reasons which I shall account for in the following pages, I have used Fuchs-Rosenthal's counting chamber. In the first instance, the daily clinical work in an otologic hospital clinic has given considerable training in using this counting chamber, which originally was intended for the counting of cells in the cerebrospinal fluid; the training with a certain counting chamber makes it difficult to adapt oneself to other

systems. Moreover, it is my personal experience that this counting chamber has other advantages. That Ellermann and Erlandsen have obtained so small a mean error as 4.96 per cent when counting 150 leucocytes, is doubtless not due to the fact that they used a dry preparation and not a moist chamber. The same result must be achieved, whether the preparation is counted in a moist or a dried condition, provided the distribution of the leucocytes is made with equal accuracy, and errors are not introduced, such as overlooking leucocytes on account of deficient lowering into the chamber, provided the cover glass is correctly placed, and provided that the counting chamber really is of the volume with which is reckoned, i. e., is exact in its measures. The comparatively low mean error in this work must be sought partly in the fact that the leucocytes in Fuchs-Rosenthal's counting chamber are spread over a rather extensive area and are counted in a fairly large field, in contradistinction to what is the case in several of the other counting chambers, e. g., the original Thoma-Zeiss counting chamber, and, partly in the very manner in which the leucocytes are spread in the area in question. In the technic generally used for blowing out the fluid in the different counting chambers due regard is not paid to the fact that in the very blowing out of a fluid in which many small bodies are suspended, these bodies may be flung in an arbitrary direction, more especially if air bubbles get into the drop in the counting chamber, even though such air bubbles are brought to disappear before the cover glass is placed in position. In a small counting chamber in which only a very small area is counted, it is evident that the distribution of leucocytes must be very arbitrary. In Bürker's counting chamber, and in the method stated by him, the blowing out is avoided by having the fluid drawn into the counting chamber by capillary attraction. However, in experiments with this counting chamber, I did not succeed in obtaining a distribution of the leucocytes which can equal the distribution in the method I have used.

The method is as follows:

1. After shaking of the hemolysed fluid for 40 seconds the pipette employed is emptied of this fluid down to a mark which, empirically, is placed at such a level of the pipette that the quantity blown out just reaches the edge of the counting



chamber, when Newton's rings appear after adjusting the cover glass.

II. During the filling of the counting chamber the point of the pipette is placed on the bottom of the chamber, and the blowing out is made slowly, care being taken to avoid air bubbles by holding the point of the pipette constantly within the drop. The whole column of fluid is not blown out of the pipette; the lowermost end of the column is allowed to remain in the pipette up to a mark placed a few mm. from the point of the pipette. Thus the fluid blown out into the counting chamber is a constant quantity.

III. The counting chamber is placed on a leveled glass plate which, by the aid of three adjusting screws controlled by a spirit level, is absolutely horizontal. In lieu of a glass plate it is possible, by the aid of the level, to make sure that the counting chamber is placed horizontally on a table.

IV. The fluid is distributed by spreading it in the counting chamber with an angular platinum needle, the bent part of which is linear. The spreading is made carefully and must be made in different directions.

V. The cover glass is placed in position by means of a small handle which, like a sort of hatch, is placed over the counting chamber, turning round an axis formed by one of the outer borders of the same. The cover glass in this way approaches to the horizontal plane when it touches the fluid.

VI. Waiting for five minutes until the leucocytes have settled to the bottom of the counting chamber. By using two counting chambers it is possible to count continuously, as a count together with cleaning of the counting chamber by rinsing and drying, and another filling takes four to five minutes.

VII. Employing a mechanical stage and a magnification of about 350 times, two transverse belts, i. e., about 200 cells, are counted in a number of leucocytes of about 10,000. Each transverse belt contains 16 small squares. All leucocytes in each little square are counted, including the cells in each square touching the lower and the right limit; it must be kept in mind that for each fourth little square a triple line is found, in which the middle line represents the limit.

Supposing that the number of cells counted is T, the number of leucocytes per cubic millimeter, with a dilution of 1:20,

$$\text{equals } \frac{T \cdot 20}{2/10 \cdot \frac{1}{4} \cdot \frac{1}{4} \cdot 32} = T \cdot 50, \text{ the area of the}$$
  
small square being  $\frac{1}{4}$  mm.

Thus, the number of leucocytes is the number of cells counted in two transverse belts multiplied by the quotient 50.

In order to determine the mean error it is necessary to procure a blood mixture that is constant. For the making of this standard blood, the following mode of procedure was used: Through a thick Wassermann canula quickly streaming blood is gathered from a vein in the arm in a measuring glass, in which is placed a 3 per cent dilution of citrate of soda, in the proportion of one of citrate of soda to five of blood; after shaking, the mixture is immediately poured into a glass bottle with a broad flat neck and a tight glass—or gum—stopper, in which are found three or four glass pearls. The bottle is then shaken for nearly one minute and then kept at a temperature of about  $+3^{\circ}$  C. Even if a few drops of formalin are added, I have preferred every day, when working with ten counts for the calculation of the mean error, to procure fresh standard blood. After having placed a glass pearl in Ellermann and Erlandsen's mixing glass and after having pipetted off 475 ccm. of acetic acid tinged with methyl violet, the standard blood is shaken for nearly one minute before pipetting off the 25 ccm. of the mixture. This shaking of the standard blood is made in the horizontal plane, taking care to avoid rotatory movements, as the leucocytes may be unequally distributed, by virtue of the centrifugal force. If perpendicular movements are made during the shaking, it will not be possible to avoid air bubbles in the standard blood. In the pipetting off, which should be performed quickly, the point of the pipette is placed between the bottom and the surface of the standard blood. After drawing off the blood, the outside of the pipette is carefully dried.

In the tables in my dissertation, pages 31 to 35, the mean error is determined for averages of 129, 102, 92 and 47; in

the first three tables, two transverse belts are counted in Fuchs-Rosenthal's counting chamber, containing 258, 204 and 184 cells, respectively; in the last table, three transverse belts are counted, showing 141 cells on an average. In order to calculate the mean error the number of cells found in two and three transverse belts, respectively, has been reduced to figures corresponding to one transverse belt in Fuchs-Rosenthal's counting chamber, by dividing by 2 and 3, and this figure is exactly the number of leucocytes for which the determination of the mean error is to be made after multiplication by 100.

In each group eight ten-counts are made. In each of these counts the average error was determined as the square root of the sum of the squares of the average deviations divided by 9, as will appear from the tables. The individual mean errors are then calculated as percentages of the average.

The mean error of eight ten-counts has been calculated from the individual average errors according to the formula:

$$m^2 = \frac{(n_1 + 1)m_1^2 + (n_2 + 1)m_2^2 + \dots (np + 1)m^2}{n + p}$$

where  $n_1$   $n_2$   $np$  represent the number of counts in each of the rows counted, which in the tables below is 10;  $p$  is the number of rows counted, which in this instance is 8;  $m_1$ ,  $m_2$ , etc., represent the individual average errors and  $n$  the total number of counts, which are here 80. As in all tables 8 ten-counts have been made, the formula in each table will be

$$m^2 = \frac{(10 - 1) (m_1^2 + m_2^2 + m_3^2 \dots)}{80 - 8}$$

The mean error thus is the square root of the sum of the squares of the average errors divided by 8.

Pursuant to the exponential law of errors the absolute figure will not deviate from the value arrived at by observation more than either plus or minus the mean error multiplied by 4; only 0.3 per cent of the values found fall between 3 and 4 times the mean error, 95 per cent fall within 2 times the mean error, 68 per cent within one time the mean error.

In the present work this means that, excluding four times the mean error as a very slight possibility, and reckoning with

three times the mean error in a number of leucocytes of 12,900 ( $m = 4.95$ ), we may obtain deviations of 1,916 leucocytes in each direction, or 14,816 upwards, 10,984 downwards.

It is of special interest to examine the range of error in a leucocyte count which is close to the limit between the normal state and leucocytosis, a limit which, according to existing works on the subject (especially Ellermann and Erlandsen), may be fixed at 10,000. For this purpose ten-count No. 2\* may be used where, with an average of 102, a mean error of 5.24 is found, i. e., when reckoning with three times the mean error, deviations of plus or minus 1,603 leucocytes may be found, thus the upward number is 11,800. Pursuant to these facts we must, therefore, reckon with a "neutral zone" between 10,000 and 11,800, or practically 12,000, where the values found may mean an increased number of leucocytes, but also may mean an error in counting.

From ten-count No. 4,† it will be seen that, although the deviations from the average in the different rows are not very great, the mean error is 10.45, on account of the low average value, 47, which, when computing the mean error, involves a multiplication by more than 2.

These conditions may be improved upon by counting over a larger area. By counting 6 transverse belts with equally low averages, I have been able to bring the mean error down to 6.5 per cent of the average value. Therefore, I count 6 transverse belts in leucocyte counts not exceeding 5,000.

#### B. TECHNIC IN DIFFERENTIAL COUNTS.

In Gustav Jørgensen's experimental investigations on the variations in the numbers of leucocytes following upon injections with strophanthin, the mean error is computed to 1.5 per cent for polynuclears which, just as the mononuclears, were enumerated in the dry preparation simultaneously with the total count; for mononuclears, the mean error was found to be 4.6 per cent: the quantitative proportion between the two groups was on an average as 3 to 1.

In the present work it has been necessary to make differential counts in stained films, and the task, therefore, has been

\*See my dissertation, page 32. †L. c., page 34.

(1) To apply a mode of spreading giving as even a distribution as possible.

(2) To apply a staining method which clearly permits the differentiation of the different groups of cells.

(3) To determine the average error for the different groups of cells in order to judge of a possible change in the blood picture within the separate groups.

The original method consists in placing a drop of blood in the middle line near one end of a completely clean slide. The drop of blood is now spread in the direction of the longitudinal axis of the slide by means of another slide with a sharp cut edge, placed at an angle of forty-five; this is done by drawing the drop, which lies in the angle, by means of this obliquely placed slide, along the preparation slide. An error is introduced by this method, arising from the fact that the polynuclears and the monocytes, which are considerably larger than the lymphocytes, will slip away under the edge of the oblique glass to a smaller degree than the latter, and are dragged along, by which they gradually are whirled towards the sides of the spread area or become placed in the marginal portion of the preparation.

As soon as this fact was realized, the corners of the obliquely placed slide were cut off, by which the displacement of the polynuclears towards the edges was decreased; however, by this modification the polynuclears were not prevented from being dragged along, and thus gave rise to an unequal distribution. This even might reverse the formula from the polynuclear type to the mononuclear one, and vice versa, depending upon whether the counting was undertaken in one part or the other of the preparation. The error is greatly diminished by counting, partly, widely separated areas in the preparation, and partly, in zigzags from edge to edge rectangularly on the longitudinal axis and, after counting one or two adjacent squares, again back to the other edge. It is scarcely to this error of distribution alone that we may ascribe the conflicting results found in previous publications undertaking to find possible deviations in the blood picture under certain conditions, e. g., in digestive leucocytosis. Nor can I believe that

this counting error is responsible for the very great variations in the leucocyte formula found within the same individual in children of different ages, aside from the great variations found in different individuals of the same age. Similar conditions are found in this work (cf. the chapter on the blood picture in normal children) by a method in which these sources of error are avoided and which is as follows:

A coverslip measuring 24 by 32 mm. is used. A little split of wood is glued along the short edge of the glass, overhanging it a little with its ends, in order to form a sort of handle. A drop of blood is put upon the "object glass," somewhat towards the left, and the cover-slip described is laid down upon it. At the moment the drop now is spreading under the coverslip, the "handle" is pulled gently to the right, by which movement the cover-slip carries the blood all over the surface of the object glass, distributing the contents equally everywhere. I have made sure that the distribution is good, and that the above mentioned errors have been avoided; by counting at one or the other end of the preparation the same percentage distribution was found; also, the distribution found a few fields' breadth from the edge of the film was found to be equally good as in the center of the slide, and by counting from one edge to the other and back again, the small error found in the outermost three to four fields in the marginal portions is adjusted, fifty to seventy-five fields of vision being the rule from one side to the other. I have tried the method proposed, according to which the cover glass is placed on the drop, which spreads equally. I found this method absolutely ideal as to the distribution, but I have chiefly used the method described above, to which I have become accustomed, and the accuracy of which I shall account for in the following pages.

I have used the following staining method, which also was used by Skat Baastrup, for examining the blood picture of scarlatina, and which was adopted after some experimentation.

1. Fixation for five minutes of the air dried film of the preparation on absolute methyl alcohol.
2. Pouring off and air drying.
3. Staining for ten minutes with a fluid consisting of equal portions of Leishmann's stain and distilled water, to which are

added six to eight drops of Giemsa's staining solution for each 10 cm. of the fluid.

4. Rinsing with distilled water.

5. Differentiation in distilled water for about ten to fifteen minutes, until the preparation becomes pink.

In addition to yielding a bad distribution of the cells, a too thick smear preparation has also the disadvantage that it stains badly, and, moreover, the pink color does not appear in the differential stain. Quite decolorized areas will be found and also areas of a brownish green color, in which areas neither details in the protoplasm nor in the nuclei of the cells can be discerned with any certainty. In this method, the nucleus of the polynuclears takes a violet stain, the protoplasm of the neutrophile polynuclears show up a pale blue color with clear, fine red granules. The protoplasmic granules of the basophile polynuclears take a vivid violet stain when fixed in methyl alcohol; if fixed in Leishmann's solution, they will be dissolved by water and appear as vacuoles. The protoplasmic granules of the eosinophiles appear brick colored, the nuclei of the lymphocytes are sharply defined of a violet stain, and the narrow protoplasmic belt of these cells is of a vivid blue color. The monocytes, both the great mononuclears and the nuclei of the transitional forms, are less vividly stained than the other cells, vacuoles of the nuclei being clearly discernible, while the outlines of the nuclei are sufficiently sharp to distinguish this group from the polynuclears and the lymphocytes. In order to limit the calculations in the present work, all mononuclear cells which were not lymphocytes, and also all the mast cells—the number of which plays only a minor rôle in the total blood count—have been included in the group of monocytes. Although it is acknowledged that this mode of procedure is not fully correct, it has been followed numerous times, both in earlier and in quite recent works. Also, the myelocytes found in the blood pictures of highly febrile acute infections have been included in the fourth group under the collective designation: monocytes, although, from a morphologic point of view, this is absolutely unwarrantable. In the hematologic literature only quite a few determinations of mean errors in differential counts are found.



By applying Ellermann and Erlandsen's dry preparation method, Gustav Jörgensen obtained a distribution which appears, also in differential counts, not to be surpassed by any other method (average error for polynuclears 1.5 per cent, for mononuclears 4.6 per cent).

For the determination of mean errors in the four different groups of cells in the stained smear preparation, standard blood (citrate blood) ought, of course, not to be used, as the spreading of citrate blood, in which the coagulation of the blood is done away with, does not correspond to the conditions under which the work in the hospital is done, when spreading a drop of blood from the earlobe on the slide. Ten-counts have, therefore, been undertaken with smears taken from the same individual, one specimen after the other, care being taken that the individual is kept under the proper experimental conditions; and if, simultaneously with the differential count, the total count is determined in these smears, and it is found that this does not vary beyond the range of error, there is the greatest probability that the drops of blood examined have a constant composition as regards white cells; this mode of procedure has at the same time the advantage of being similar to that used in the clinical examinations for which it is desired to determine the mean error.

#### SUMMARY OF CHAPTER II.

I. By using the separated pipettes and mixing glasses devised by Ellermann and Erlandsen, by pipetting off a constant quantity of the fluid for each count in a Fuchs-Rosenthal's counting chamber and by spreading the fluid carefully in the leveled counting chamber, the mean error was found to be

- 4.96 per cent in an average of 129 (258 cells counted)
- 5.24 per cent in an average of 102 (204 cells counted)
- 5.85 per cent in an average of 92 (184 cells counted)
- 6.50 per cent in an average of 50 (300 cells counted in 6 transverse belts)
- 10.45 per cent in an average of 47 (when counting only in 3 transverse belts).

II. By using the cover-slip method and also a modification of the same, in which the drop is allowed to spread widely below the cover-slip before smearing, a mean error of 3.02 per

cent for the polynuclears is obtained in a count of 400 cells, of 33.23 per cent for the eosinophiles, of 8.22 per cent for the lymphocytes, and of 31.87 per cent for the monocytes.

### CHAPTER III.

#### BLOOD STUDIES IN NORMAL ADULTS (A SHORT SERIES OF INVESTIGATIONS).

In the introduction an essential part of this work is mentioned, the object of which was to determine the normal number of leucocytes in healthy adults, and as this question has been treated so elaborately, I have thought it desirable to make a small number of examinations of the blood picture in adults, in order to have, through my own examinations, a basis for deciding the blood picture in pathologic states. The samples of blood were taken at 11 a. m., as was also the rule in the examinations of normal and adenoid children. I made sure that each individual to be examined had taken only a light breakfast, poor in albumen, none of the patients being examined until 2½ to 3 hours subsequent to such a light meal. The samples were taken while the individuals were in a sitting position, after having waited quietly at least 15 minutes, care being taken to avoid psychic and static leucocytic reactions. In women I avoided taking samples from those individuals who showed the slightest sign of psychic unrest. All the persons were questioned as to possible recent febrile diseases, and they were all submitted to a general objective examination, by which I feel that I have excluded those who could not be called healthy. A total of thirty individuals, between the ages of from 18 to 49 years, were examined. In a minority of cases several examinations were made on successive days, but only on a few days, at a definite hour, i. e., at 11 a. m.

TABLE ON BLOOD EXAMINATIONS IN NORMAL ADULTS.

Age	Sex	Total	Polynu- clears	Eosino- philes	Lympho- cytes	Mono- cytes
18	F.	7900	49.5	5.0	37.5	8.0
18	F.	8000	57.7	2.3	32.3	7.7
18	F.	7500	48.9	3.3	42.4	5.4
18	M.	10200	48.2	3.8	44.2	3.8
18	M.	7300	59.5	1.5	33.8	5.2

Age	Sex	Total	Polynu- clears	Eosino- philes	Lympho- cytes	Mono- cytes
20	F.	7900	49.9	4.9	37.9	7.3
20	F.	6700	59.0	2.4	32.3	6.3
20	M.	7800	54.4	4.5	32.9	8.2
20	M.	8400	68.1	0.8	24.6	6.5
26	F.	7400	62.0	2.1	28.0	7.9
26	F.	6100	59.6	1.8	30.0	8.6
28	M.	9900	52.5	2.9	38.3	6.3
29	M.	7900	63.5	2.0	30.4	4.1
31	F.	8200	60.3	2.0	32.1	5.6
31	F.	8400	59.2	2.4	33.0	5.4
39	M.	9000	68.1	1.9	25.9	4.1
40	M.	7400	60.1	1.6	34.6	3.7
41	F.	7000	55.3	4.5	38.3	1.9
49	M.	6500	51.8	3.4	41.6	3.2
49	M.	8900	65.0	4.3	27.0	3.7
49	F.	8800	60.0	2.3	31.7	6.0
49	F.	7900	62.3	1.8	29.7	6.2
Averages:		7959	57.9	2.8	34.0	6.2

It will appear from these figures that the highest number of leucocytes in these 22 normal adults was 10,200, the lowest 6,100. The average for these 22 normal adults, who were examined at 11 a. m., was 7,959. This value is somewhat lower than that found by Ellermann and Erlandsen (8,800 at 10 a. m., 25 counts). The average for women (a total of ten) was 8,330, and for men (a total of twelve) 7,650. This is in harmony with previous investigations, as Bruhn-Fähräus, Kjer-Peterson, H. C. Gram and Lindström and Tallquist find somewhat smaller figures for men than for women. The same result was obtained by Rud, who in a total of eighteen persons, who were employed in Röntgen and radium institutes, found 4,900 leucocytes on an average in men, 5,850 in women at 11 a. m. I cannot omit to note that these figures are considerably lower than those found both by Ellermann and Erlandsen and by myself. And looking at the individual values, which lead to this low average, it appears that there were two of 2,800, 4 below 4,000 (blood from the earlobe) and 3 between 4,000 and 5,000. figures which, from my own experience in this hematologic work, must be considered as pathologically low, and I should therefore think that these low figures in reality are due to X-ray injury of the leucocyte forming tissue.

As to the proportions of the various cells, it appears that there were rather great variations in the different individuals;

the lowest for polynuclear neutrophile cells was 48.2 per cent in a woman aged 18; the highest, 68.1 per cent, in a woman aged 39. Polynuclears, 57.9 per cent; eosinophiles, 2.8 per cent; lymphocytes, 33.1 per cent; monocytes, 6.2 per cent, were the average values found.

This result is in complete disagreement with the statements found in the hematologic textbooks. Pappenheim, for instance, gives the normal value for neutrophile polynuclears in adults as 73 to 75 per cent, while Näegeli gives 65 to 70 per cent. I thought that I alone had found these discrepancies, until Rud's work drew my attention to the fact that modern Finnish hematologic studies give figures which literally coincide with my results. Thus, Appelberg states that the averages found by Bonsdorff, Terhola, Becker, Sandelin and Appelberg are the following: Neutrophile polynuclears, 56.3 per cent; eosinophiles, 3 per cent; lymphocytes, 30.5 per cent; monocytes, 6.7 per cent.

Corresponding figures found by Lindström and Tallquist are: Neutrophiles, 59 per cent; eosinophiles, 1.6 per cent; lymphocytes, 32 per cent, and monocytes, 6.5 per cent.

The 22 individuals examined were from 18 to 49 years old; 9 were between 18 and 20 years. In these 9 adults 35.3 per cent of lymphocytes were found on an average. When comparing this figure with the corresponding figures for individuals in this series between the ages of 26 and 49 years, a total of 13 individuals showing a percentage of 30.8 of lymphocytes on an average, it will be noted that the decrease in the number of lymphocytes, which is doubtless through the years of childhood (see Chapter V), is continued in the adult age, in other words, the infantile lymphocytosis is continued in a juvenile lymphocytosis, which is on the decline between the ages of 25 and 50. This result, which was arrived at here in a series of examinations which is, perhaps, too small for judgment, and which therefore no doubt ought to be controlled in a larger material, seems to agree well with the theory that the whole lymphatic system, which reaches its maximum development in childhood, undergoes a gradual atrophy in the course of the years in adults.

As to the eosinophiles, it also appears in this series of examinations that the gradual decrease which takes place in

childhood is continued in youth. Thus, with this material, the percentage of eosinophiles found in the ages of 18 to 20 years is 3.2, while in the ages between 26 and 49 it is only 2.5.

In addition to these investigations, some serial examinations on three to five successive days were undertaken, yielding the following results:

Age	Sex	Date	Total	Polynuclears	Eosinophiles	Lymphocytes	Monocytes
22	F.	5/8	7800	63.0	2.8	29.0	5.2
		6/8	8200	59.8	1.0	31.6	7.6
		7/8	7500	64.0	1.5	29.0	5.5
19	M.	5/8	6500	57.0	4.3	31.0	7.7
		6/8	8200	59.3	3.0	33.0	4.7
		7/8	6400	67.8	5.3	25.3	1.6
24	F.	9/8	8200	71.8	1.7	24.8	1.7
		10/8	8900	63.7	2.1	29.6	4.6
		11/8	8000	59.1	4.2	31.7	5.0
36	F.	9/8	8900	72.8	1.7	18.7	6.8
		10/8	6300	65.9	2.8	30.5	0.8
		11/8	7000	71.0	1.8	22.7	5.5
		12/8	8900	63.1	3.6	26.5	6.8
44	M.	8/9	8200	68.5	2.2	24.3	5.0
		9/9	9000	70.6	0.4	27.1	1.9
		10/9	8100	65.9	3.2	22.9	8.0

Considering the variation from day to day, it appears that this is rather constant, and taking the average for each individual on the third or fourth days of examination and reckoning with a mean error of 6.5 per cent, such as I have found it—by counting six transverse-belts at low averages, it will be seen that in each individual case the variation falls within  $3 \times 6.5$  per cent, or 19.5 per cent. Thus in Case 1: average value 7,833, mean error 6.5 per cent, upper limit of error in counting 9,360, lower limit 6,306. Case 2: average value 7,033, mean error 6.5 per cent, upper limit of error 8,404, lower limit 5,662. Thus it will be seen in each individual case that the figures fall within the range of error—i. e., that we do not know with certainty if a true variation in the number of leucocytes is found in the same individual in the above series of examinations.

Age	Number of leucocytes	Maximum	Minimum	Polynu- clears, per cent	Eosino- philes, per cent	Lympho- cytes, per cent
1- 2						
2- 3	9400	10000	7500	36	4	53
3- 4	8400	10000	7200	36.5	5	48.5
4- 5 }	8500			46	3	43
5- 6 }						
6- 7						
7- 8 }	7900	10000	6000	52	6	34
8- 9 }						
9-10						
10-11						
11-12						
12-13 }	7500	7000	6500	60	2	30
13-14 }						
14-15						

## CARSTANJEN.

Age	Polynuclears, per cent	Eosinophiles, per cent	Lymphocytes, per cent	
2-3	48.2	3.9	38.5	Total values not given.
3-4	52.6	5.7	33.2	
4-5	61	6.3	25.1	
5-6	55.4	6.2	31	
6-7	57.8	3.3	30.3	
7-8	60.9	3.7	27.9	
8-9	58.6	5.5	28	
9-10	57	5.5	28.4	
10-11	51.9	7.3	33	
11-12	60.7	3.1	28.3	
12-13	53.8	3.6	33.3	
13-14	62.8	4.2	25.9	
14-15	56.5	5.9	28.2	

## RABINOWITZ:

Age	Polynu- clears, per cent	Eosino- philes, per cent	Lympho- cytes, per cent	Lympho- cytes	Maxi- mum	Mini- mum
1-2	6500	30	3.6	61	3965	5656
2-3	7400	35	5.8	55.5	4080	5490
3-4	6400	35.5	6.2	52.5	3360	4562
4-5	6600	38	6.5	52.0	3410	4191
5-6	7100	50.5	3.0	42.5	3020	3844
6-7	6800	49.5	5.4	39.0	2650	3671
7-8	6300	47.5	6.4	36.0	2275	3167
8-9	6100	50.0	5.6	36.5	2240	3306
9-10	6200	51.5	5.4	36.0	2220	2788
10-11	5900	55	4.4	34.5	2020	3903
11-12	6800	53	7.5	34.5	2340	2912
12-13	6300	56.5	4.8	31.5	1917	2664
13-14	6900	58	4.3	31.0	2135	2944
14-15	7000	65.5	3.8	25.0	1750	2268
						880

The last table gives the averages for four to six children of both sexes of different ages; no difference was found in the blood picture in the two sexes. As to the total values, which in the table above are given as averages, a difference between the maximum and the minimum was found in different children within the same age, sometimes amounting to more than 100 per cent—e. g., in the age between 9 and 10 years, a maximum of 10,200, a minimum of 4,200. The highest number of leucocytes found was 10,400, the lowest 4,200. It will be seen from the table how the lymphocytes are predominant in the younger ages, rather evenly decreasing from 61 per cent to 25 per cent, and how the polynuclear neutrophils are in an inverse ratio, increasing rather evenly from 30 per cent to 6.5 per cent, while the values for the eosino-

philes do not follow any regular curve, but, in the majority of cases, range between 4 and 6 per cent. In the latter part of the above table it will be seen that very great variations are found within the individual ages within a separate kind of cells, such as lymphocytes, and, of course, the same holds good in regard to the polynuclears, a fact which has been elaborately established in Rabinowitz's work and therefore is not especially worked out here. It appears that the difference between the maximum and the minimum values is often more than 100 per cent, and the tables given in this treatise on the percentage contents show that this may also vary considerably within the individual age group, although never reaching so great differences in values as found in the absolute figures. To mention a few instances: In the age group of 2 to 3 years, 48 to 61 per cent; 3 to 4 years, 46 to 61 per cent; 5 to 6 years, 37 to 51 per cent; 6 to 7 years, 33 to 48 per cent; 9 to 10 years, 33 to 45 per cent; 11 to 12 years, even 20 to 45 per cent, corresponding variations being thus also found in the group of the neutrophile polynuclears.

As a basis of the studies of the blood picture in children suffering from different infections, I have considered it to be of importance to examine the blood picture of normal children by means of the technic described. From the following tables it will appear that just as was the case in previous works, great variations have been found within the same age group, both in the total count and in the differential count of the blood cells. The subjects to be experimented upon had to be selected with the greatest care. In other words, it was rather difficult to find what in the strictest sense might be called perfectly healthy children, especially as these examinations were made in winter, and it appeared that some of the children, who were inmates of infant homes, were catarrhal, that others suffered from furunculosis, others, again, from adenitis, etc. Moreover, it was necessary to determine what children had recently had fever, for under such circumstances a possible postinfectious lymphocytosis must be taken into consideration. For these reasons the number of normal children within the different age groups is not as great as it might be, more especially as a comparison is to be made in the following



pages between the blood picture found in normal children and that of adenoid children. In the publications referred to, the authors have not made serial examinations from day to day, nor have such been undertaken in the present work except in a few instances. In these few cases, the composition of the blood appeared to vary without any traceable reason, so that, for instance, one day 38 per cent of lymphocytes, the next day 52 per cent, were found in the same child. Apart from the fact that it would be an interesting task to undertake serial examinations of children's blood, the substantiation of these variations, which are more or less obscure, but which, nevertheless, seem to be established, would give a somewhat uncertain character to the determination of the blood count in normal children within the various age groups. On the other hand, it appears, as is also evidenced in the tables below, that by determining the average of the counts of say, three to four children of each group, we get a fairly evenly falling curve for the lymphocytes, however, as might be expected, with certain variations in increase or decrease from one age to the other, and a corresponding inverse increase in the polynuclears, also with irregularities in the increase, which presumably would have been diminished if each group had comprised a greater number of children. As to the eosinophiles, no definite law has been found for the increase or the decrease within the different ages beyond the fact that there is a higher percentage value before the age of seven years than after this age, which is in full harmony with what was found by all other investigators.

The group of the monocytes shows no variation, when comparing the younger and the older age groups. The average total count in the ages of from three to fourteen years in a total of 47 children was found to be 7,674. The average percentage value of the polynuclears within the different groups was computed by dividing the average total value for each group into the average partial value for the polynuclears in each group. It shows a fairly even increase from 42.40 per cent, at the age of three years, to 55.61 at the age of six years, after which it keeps near 50 per cent until the age of ten is reached, when it increases to 55 per cent, at the age of eleven years; again it decreases to nearly 50 per cent at the age of

twelve or thirteen years, and increases again to 56.69 per cent at the age of fourteen. Inversely, the lymphocytes decrease rather evenly from 47.32 per cent at the age of three to 36.54 per cent at the age of six. From seven to eleven years the percentage of lymphocytes is near 40, similar values being found again at the ages of twelve and thirteen years, while at eleven and fourteen years values near 35 per cent are seen. Only by estimating the figures throughout the whole period from three to fourteen years are the increase substantiated by many investigators in the polynuclears and the decrease in the lymphocytes found.

STUDIES ON THE VARIATIONS IN THE NUMBER OF LEUCOCYTES  
FROM DAY TO DAY IN NORMAL CHILDREN.

As stated in the preceding chapters, what previous authors, especially A. v. Bonsdorff, have found has been confirmed—i. e., that variations in the numbers of leucocytes are found from day to day in adults both in the total number and in the differential count—variations which partly fall within the mean error, but which also, as far as some values are concerned, fall outside the limit of error. It was to be expected, and perhaps to a still higher degree, that variations in the blood picture of normal children must also be found, more especially considering that the leucocyte formula of children shows only a slight difference between the numbers of lymphocytes and leucocytes in the individual subject. In the determination of the mean error alone, it is evident that, in certain cases, now a lymphocytic formula, now a leucocytic one, will appear with blood of a constant composition, when the values for these kinds of cells approach one another. Furthermore, we must take into consideration that quite mild and often clinically nontraceable infectious conditions, such as light catarrhs, may raise the number of leucocytes without producing a definite leucocytosis, and that such a slight rise in the leucocytes is essentially due to polynuclear leucocytes. From these very reasons, it may easily be foreseen that the blood picture of an apparently normal child may be submitted to rather considerable variations. In the comparatively small number of examinations I have made in this respect, this supposition was also corroborated.

Age	Sex	Date	Total Number	Percentage of different cells:			
				Polynu- clears	Eosino- philes	Lympho- cytes	Mono- cytes
7	M.	5/4	10800	52.9	5.7	29.6	11.8
		6/4	5800	40.4	5.0	52.3	2.3
		7/4	9500	54.9	2.0	40.0	4.1
9	F.	2/11	5900	47.8	2.7	41.1	8.4
		3/11	5700	55.1	3.0	39.9	2.0
8	F.	2/11	5200	35.0	2.4	54.0	8.6
		3/11	7700	52.0	1.3	41.8	4.6
		3/11	8600	50.1	4.7	39.3	5.9
6	M.	4/11	8300	46.1	5.9	42.9	1.1
		5/11	7300	37.8	6.6	48.8	6.8
		6/11	9500	50.9	6.5	38.6	4.9
		8/11	5300	39.7	4.3	54.3	1.7
		10/11	9600	40.8	5.5	51.6	2.1
		11/11	8000	48.8	5.9	39.0	6.2

If we look at the individual totals of the same child in this small series of examinations, it is quite evident that the differences found in Case 1 and in Case 4 are so great that they may be said with certainty to fall outside the limit of error; thus in Case 1, on April 5th, the count shows 10,800; on April 6th, 5,800; in Case 4 the counts are: on November 11th, 9,500; on November 8th, 5,300; on November 10th, 9,600. These variations in the numbers were not accompanied by any regular changes in the proportions of the various cells; the transient increase from one day to the other might be due to light degrees of catarrhal states, but, if this was the case, a corresponding increase of the polynuclear group would be expected. While this perhaps might seem to hold for Case 1, it does not hold good of Case 4, where, on November 6th and November 10th, with nearly an identical number of leucocytes, a percentage of 50.9 of polynuclears was found on the first day and of 40.8 on the last day. So great variations in the differential blood cell count are not found within the same individual in adults (see Chapter VI), where the variations in the polynuclears—reckoning with three times the mean error, or three times 3.02 per cent = 9.06 per cent, or about 10 per cent—on the whole, appear to fall within the limit of error. In adults, however, in Cases 1 and 2 for instance, variations were found which, although far from reaching the great differences found in children stated, nevertheless amounted to just above three times the mean error, as will be seen from the following calculation: Case 2 (average value determined to be 62.4), 70.8

thus deviates from the average more than three times the mean error, which would be 9.06 per cent of 62.4, or approximately 10 per cent of  $62.4 = 68.64$ , while, in reality, on August 7th, 8 per cent was found. In Case 3, 71.8 per cent was found on August 9th, while 71.2 per cent should be the extreme limit of error. In the other cases the figures vary within the limit of error; it is seen that also those cases in which the figures fall within the range of error show rather great differences in value, so that—if these were to be considered as counting errors—I must constantly have been very unfortunate in my counts, getting the highest permissible deviations from the average pursuant to the exponential law of error. This, however, does not at all agree with the exponential law of error, according to which even 95 per cent of the values found fall within twice the average error and 67 per cent fall within once the average error. Accordingly, it may be inferred that both in adults, and especially in children, variations in the blood count exist from one day to the other. In the present work I have not tried to find the cause of this variation. These results do not agree with the small diurnal variations found in the composition of the blood from 6 a. m. to 7 p. m., variations which were seen quite possibly to fall within the limit of error. The majority of the patients examined for diurnal variations were in bed; a single case, who was not confined to bed, remained quiet in the hospital ward, while the normal adults and normal children were individuals who were examined under uniform conditions, but who were out of bed and out of doors and thus lived under conditions highly different from those of the patients in a hospital. Without being able to say anything certain as to causation, it seems reasonable to suppose that the greater variations found in the outpatients are just due to the uncontrolled conditions of the former category, as some of the patients perhaps have walked much and thus may have incited the leucocyte forming tissues by muscular movements and exercise. Such outpatients cannot be compared with those in the hospital, because in the one case it was a question of examinations within the same day, in the other case of examinations made from one day to the other. This problem might perhaps afford an interesting task for future investigation.

## CHAPTER V.

## THE BLOOD PICTURE OF CHILDREN WITH ADENOID GROWTHS.

The etiology of adenoid growths has of late years been the subject of much discussion, divergent views having been advanced both within the circle of pediatricians and also between pediatricians and otolaryngologists. When Czerny twenty years ago established the clinical type of exudative lymphatic diathesis he had through many years clinically studied a series of small patients in the hospital ward, who previously had been treated according to their individual symptoms, at times for eczema, strophulus, urticaria, etc., at other times for conditions of the mucous membranes, such as frequent anginas, laryngitis, trachitis, bronchitis, etc. Furthermore, these children differed from the other children in a changed state of nutrition, two types being especially found: children with increased panniculus adiposus and lean erethic children. This clinical type has gradually become acknowledged by a great number of clinicians all over the world, but there is still a great group of pediatricians who do not quite acknowledge this type, or, at any rate, who delimit it considerably, in contradistinction to other clinicians who go so far as to reckon the lymphatic hyperplasia found even in older children as a feature in the exudative lymphatic diathesis, provided that, in addition to the objective symptom of the diathesis, only a single one of the other symptoms is found in the anamnesis or the objective examination. Czerny's clinical type: exudative lymphatic diathesis concerned children in their first years, and although there are clinicians who go far beyond the original scope of this category of children, the general view is that, in older children with adenoid growths and tonsillar hypertrophia, the connection with the exudative lymphatic diathesis cannot be maintained, as not even the main features of the syndrome can be found in the majority of the cases.

For some time the changed blood picture played a part in the syndrome of the exudative lymphatic diathesis. On the basis of the view that this diathesis came under the category of lymphatism—a condition which in itself exists physiologically in the child, but in children should be considered a pathologic lymphatism—and perhaps also under the impression of

an "anaphylactic" state in the exudative lymphatic diathesis. Langst in and Putzig, and also Rosenstern, were of opinion that the exudative lymphatic diathesis was characterized by an increased number of eosinophile cells and lymphocytes. These authors thought that these two kinds of cells were found in increased numbers, even if no dermal affections were present, while Aschenheim and Kroll—the former in a treatise in which he criticizes Putzig's work—maintain that the changed blood picture is closely connected with the eczemas and disappears simultaneously with them. Kroll did not find any changed blood picture in doubtless cases of exudative lymphatic diathesis showing affections of the mucous membranes but no skin disease. That eosinophilia, and in certain cases also relative lymphocytosis, are found in a great number of skin affections which need not belong to the exudative lymphatic diathesis, but which, at any rate, may be features of this disease, has been shown by Lauener. He set himself the task to examine the blood picture in children suffering from these various skin diseases; however, he did not examine the blood picture after the skin affections had disappeared, so that he could not decide whether the changed blood picture is a secondary or a coordinate phenomenon with the skin diseases. His findings in chronic eczema of children were:

Case	Age	Polynuclears, per cent	Eosinophiles, per cent	Lymphocytes, per cent
1	7/12	33	9	53
1	1	34	10	49
1	1	15	11	73
1	1	37	4.5	55
1	1	26	15	54
1	1½	54	12	31
1	2	21	18	56
1	4	13	9	71
1	6	43	5	45

In some cases a slight increase of the numbers of leucocytes was found (12-13,000). In the above table eosinophilia is, at any rate, found in seven out of the nine patients, and in some cases—especially in Cases 3, 5, 7 and 8—it seems as if the contents of lymphocytes in relation to the total number of leucocytes is greater than normal.

Moreover, in two cases out of four of an acute child's eczema, Lauener found a marked increase of the eosinophile

cells (9.5 per cent and 13 per cent), but no certain increase of the lymphocytes. In three children, aged four, suffering from urticaria, 8, 3 and 4.5 per cent of eosinophile cells, respectively, were found without lymphocytosis; in eight children, from 1½ to 4 years, suffering from strophulus, an eosinophilia of a very considerable degree was found in one case only—i. e., 30 per cent, the two next values being 8.5 per cent and 7 per cent; in the other preparations no increase of the eosinophiles was found, and in most cases there was no increase of the lymphocytes, while four cases showed values which, compared with the proportion of lymphocytes in the blood of normal children, may indicate a lymphocytosis, similar values being, however, also found in exceptional cases in the examinations of normal children made by D. Rabinowitz. Three children with scrofulous eczema had an increase of eosinophiles (11.14 and 6.5 per cent) but always in connection with an increase of polynuclears.

According to the literature on the subject, it seems to be a very common, although not constant, phenomenon that eosinophilia is connected with a number of skin diseases which may be features of the exudative lymphatic diathesis, but the presence of which need not indicate that the child suffers from exudative lymphatic diathesis. Lymphocytosis seems much less constant, in reality so inconstant that it is hardly possible to consider it as a characteristic feature of these diseases.

No definite conclusion as to whether eosinophilia (and lymphocytosis) is connected with the eczemas has, as yet, been determined, the above mentioned discussion having not yet given rise to new publications on this subject. In the present work it will not be possible to contribute in any way to the solution of this question, for the children with adenoid growths, the blood count of whom has been determined, did not show any dermal symptoms and did not, as far as could be ascertained through a careful anamnestic and objective examination, suffer from exudative lymphatic diathesis. Nor do the comparisons between the blood picture of adenoid children and that of normal children made in this work help to solve the question of the relation of adenoid children to the exudative lymphatic diathesis. In the present material no children with

exudative symptoms are found—e. g., eczemas, strophulus, laryngitis, trachitis, asthma, etc.

The question of the blood picture found in connection with different forms of intestinal worms has, of late years, been very much discussed, without an agreed view. This is due, partly, to the fact that the information in hand shows high variations under apparently uniform experimental conditions, and, partly, that the various authors did not always fully realize whether the patient examined suffered from an affection due to a great many worms in the intestines, or only to a small number as chance inhabitants of the intestines. When it is considered that the majority of apparently quite healthy individuals harbor *tricocephalus dispar* as a casual parasite in their intestines, however, to such a small degree that it does not affect the general state of health of the individual, and that a still greater number of persons have *oxyuris* harmless parasites in their intestines, it will be understood that an attempt to exclude individuals in whom intestinal worms are present from hematologic examinations in the daily clinical work would be quite unreasonable. Thus, C. Jørgensen found *tricocephalus dispar*, or rather its eggs, in the intestines of 60 per cent of his total material of adults and children, in 67 per cent of the patients under 30 years of age, in 55 per cent of the patients above 30 years, while Sven Müller found *oxyuris* in 57 per cent, and in children below 15 years, in 62 per cent.

Notwithstanding these facts, Naegeli reckons with eosinophilia in patients with *oxyuris* and *tricocephalus*; and also—as most other authors—in patients with *ascaris lumbricoides*, *tenia saginata* et *solium*, *anchylostomum duodenale*, *echinococcus* and *trichina spiralis*. In several of these forms of helminthiasis a disappearance of the eosinophiles has been observed at the stage where the patient becomes seriously anemic and where the decrease of eosinophiles at the same time should indicate a decrease in the functions of the bone marrow. As to the two first mentioned groups, very few and highly unreliable accounts are at hand. Thus, Bucklers found a moderate increase of the eosinophiles in one-sixth of the cases of *oxyuriasis*, when it was a question of a real disease and not a casual finding, while French and Boycott contest the occurrence of eosinophilia in this disease, as in 27 cases they did find any



increase (2.1 per cent on an average, of eosinophile cells). Grek and Reichenstein, in half of their patients with tenia (36 out of 72), found more than 5 per cent of eosinophiles, while Ragosa and Limasset found eosinophilia in one-third of their cases. Besides the eosinophilia it is the rule to find a decrease in the total number and a relative lymphocytosis. In cases of *anchylostomum duodenale*, Bloch, Bucklers, Bruns and Liefmann and Mäkel found very high figures (15-20-40 per cent) for the eosinophiles; in carriers of *echinococcus* Ramsey found up to 28 per cent, Memmi up to 20 per cent, Sabrazès up to 17 per cent, Seligmann and Dudgeon even up to 57 per cent of eosinophile cells. In trichinosis, also, eosinophilia plays a part as a help in diagnosis; however, it is not found during the first two weeks of the infection and, in mild cases of trichinosis it is moderate, while the eosinophiles disappear almost completely in fatal cases. This disease affords a special opportunity for studying eosinophilia localized to the affected tissue.

Altogether, in a number of various forms of helminthiasis we shall have to reckon with an eosinophilia and a lymphocytosis (in trichinosis, however, the eosinophilia is combined with a neutrophile leucocytosis). These factors, however, do not to any greater extent interfere with the daily clinical examinations because, in the first place, eosinophilia is not found in connection with the usually occurring forms of helminthiasis (*oxyuris* and *tricocephalus*), except if they are found in so great quantities as to give noticeable symptoms, and even then only in a fraction of the cases. In the second place, the great tenia are not usually found in the intestines of children and adults and, when found, as a rule give rise to specific symptoms. Third: In the daily clinical work, one may practically disregard those forms of helminthiasis which constantly and to a great extent yield an increase of the eosinophile cells (such as those caused by *anchylostomum duodenale*, and, especially, by *echinococci* and *trichinae*).

Nevertheless, it is, of course, necessary to reckon with a source of error just from this quarter, when, as in certain sections of the present work, it is desired to examine the importance of the quantitative relation of the eosinophile cells (see the chapters on adenoid growths and on scarlatina).

THE BLOOD-PICTURE OF NORMAL CHILDREN  
OF FROM 3 TO 15 YEARS OF AGE.

Age		Total values for leucocytes	PERCENTAGE				PARTIAL VALUES			
			Polynuclears	Eosinophiles	Lymphocytes	Monocytes	Polynuclears	Eosinophiles	Lymphocytes	Monocytes
3	G. S.	5500	50.8	1.0	38.4	9.8	2794	55	2112	539
	H. H.	9800	40.7	3.4	50.9	5.0	3989	333	4988	490
	S. H.	7500	46.4	4.5	45.0	4.1	3480	338	3375	308
	F. F.	7900	36.3	6.6	50.1	7.0	2868	521	3958	553
							13131	1247	14433	1890
							3233	312	3608	472
		7625	Average total value.				42.4	4.09	47.32	6.19
4	E. P.	8400	50.7	4.0	40.0	5.3	4259	336	3360	445
	S. T.	8200	54.2	6.8	35.4	3.6	4444	558	2903	295
	R. S.	6700	40.4	4.1	48.5	7.0	2707	275	3250	469
	A. F.	5800	44.9	5.0	45.2	4.9	2604	290	2622	284
	J. J.	7000	46.8	6.6	40.5	6.1	3276	462	2835	427
							17290	1921	14970	1920
							3458	384	2994	384
		7220	Average total value.				47.89	5.32	41.47	5.32
5	R. H.	11500	49.1	3.3	43.2	4.4	5647	380	4968	506
	G. H.	5800	50.1	6.2	38.1	5.6	2906	360	2210	325
	C. P.	6100	44.1	7.0	42.2	6.7	2690	427	2574	409
							11243	1167	9752	1240
							3748	389	3251	413
		7801	Average total value.				48.05	4.99	41.67	5.29
6	M. H.	6800	60.1	2.8	33.1	4.0	4087	190	2251	272
	D. P.	9500	54.8	2.2	38.2	4.8	5206	209	3629	456
	G. J.	8100	50.2	2.2	43.6	4.0	4066	178	3532	324
	Aa. N.	8400	58.1	6.7	30.6	4.6	4880	563	2570	386
							18239	1140	11982	1438
							4560	285	2996	359
		8200	Average total value.				55.61	3.48	36.54	4.37
7	S. A.	10800	45.2	7.8	41.2	5.8	4882	842	4450	626
	Ch. H.	8500	53.7	5.5	34.8	6.0	4565	468	2958	510
	Chr. P.	7900	51.4	4.1	37.4	7.1	4061	324	2955	561
							13508	1634	10363	1697
							4503	545	3454	566
		9068	Average total value.							
8	S. A. H.	5800	47.8	3.4	44.8	4.0	2772	197	2598	232
	C. C.	9100	50.9	5.2	40.7	3.2	4632	473	3704	291
	J. C.	7500	52.7	3.4	40.9	3.0	3953	255	3068	225
	A. N.	7400	41.8	3.0	46.8	8.4	3093	222	3463	622
							14450	1147	12833	1370
							3612	287	3208	342
		7449	Average total value.				48.49	3.85	43.07	4.59

THE BLOOD-PICTURE OF NORMAL CHILDREN  
OF FROM 3 TO 15 YEARS OF AGE—Continued.

Age		PERCENTAGE					PARTIAL VALUES			
		Total values for leucocytes	Polynuclears	Eosinophiles	Lymphocytes	Monocytes	Polynuclears	Eosinophiles	Lymphocytes	Monocytes
9	E. H.	9000	60.6	3.8	30.4	5.2	5454	342	2736	468
	E. W.	8100	48.8	3.7	40.3	7.2	3953	300	3264	583
	C. K.	5900	46.1	3.5	41.7	8.7	2720	207	2460	513
							12127	849	8460	1564
							4042	283	2820	521
		7666	Average total value.				52.73	3.69	36.79	6.79
10	J. S.	7000	32.4	1.5	63.1	3.0	2268	105	4415	210
	R. Chr.	4800	48.6	2.4	39.4	9.6	2333	115	1891	461
	R. H.	7000	60.6	4.5	30.9	4.0	4242	315	2163	280
	B. O.	11700	51.1	1.9	43.7	3.3	5979	222	5113	386
							14822	757	13584	1337
		7624	Average total value.				3705	189	3396	334
11	V. J.	12600	54.6	4.1	35.1	6.2	6880	517	4423	781
	K. J.	5900	60.5	1.7	32.3	5.5	3570	100	1906	325
	A. H.	7200	51.9	5.9	37.3	4.9	3737	425	2686	353
							14187	1042	9015	1459
							4729	347	3005	487
		8568	Average total value.				55.20	4.05	35.07	5.68
12	E. H.	9100	49.8	8.0	35.0	7.2	4532	728	3185	655
	A. R.	7100	44.0	6.2	40.8	9.0	3124	440	2897	639
	E. S.	8800	39.3	4.2	50.0	6.5	3458	370	4400	572
	H. Chr.	8500	59.8	1.9	31.0	7.3	5083	162	2635	621
	H. S.	6500	48.7	3.6	46.1	1.6	3166	234	2997	104
							19363	1934	16114	2591
		8000	Average total value.				3872	387	3223	518
13	L. P.	7800	56.0	1.9	37.8	4.3	4368	148	2948	335
	W. C.	6600	50.9	0.7	40.5	7.9	3359	46	2673	521
	A. Chr.	6100	39.9	3.1	50.8	6.2	2434	189	3099	378
	G. S.	6800	56.1	3.9	35.8	4.2	3815	265	2434	286
	H. A.	6400	46.4	2.6	45.1	5.9	2967	166	2886	378
							3631	374	2387	408
							20574	1188	16427	2306
		6749	Average total value.				3429	198	2738	384
14	K. O.	9100	60.9	1.7	32.8	4.6	5542	155	2985	419
	E. H.	4800	58.5	4.8	30.6	6.1	2808	230	1469	293
	A. J.	6100	49.0	3.4	40.2	7.4	2989	207	2452	451
							11339	592	6906	1663
							3779	197	2302	388
		6666	Average total value.				56.69	2.96	34.53	5.82
15	O. B.	7100	63.0	5.1	27.2	4.7	4473	362	1931	334
	J. S.	7000	58.8	2.0	36.2	3.0	4116	140	2534	210
	A. H.	5300	51.2	3.4	40.1	5.3	2714	180	2125	281
	A. J.	11300	60.9	5.8	30.3	3.0	6882	655	3424	339
	P. S.	5500	51.0	2.8	40.0	6.2	2805	154	2200	341
							20990	1491	12214	1505
		7240	Average total value.				4198	298	2443	301
							57.98	4.12	33.74	4.16

## THE BLOOD PICTURE OF CHILDREN WITH ADENOID GROWTHS.

Age	Case-book No.	PERCENTAGE					PARTIAL VALUES			
		Total Number of leucocytes	Polynuclears	Eosinophiles	Lymphocytes	Monocytes	Polynuclears	Eosinophiles	Lymphocytes	Monocytes
3	1135	6900	46.3	4.4	42.5	6.8	3195	304	2933	469
	1318	9100	47.2	3.3	43.2	6.3	4295	300	3931	573
	2256	11500	41.3	4.0	49.7	15.0	4750	460	5716	575
	497	7800	44.0	7.3	45.1	3.6	3432	569	3518	281
		8000	36.3	6.6	50.1	7.0	2904	528	4008	560
							18576	2161	20106	2458
		8660	Average.				3715	432	4021	492
	1026	10800	45.4	8.2	42.4	4.0	4903	886	4579	432
		6100	36.4	6.1	53.5	4.0	2220	372	3264	244
	1029	6900	48.7	0.7	47.8	2.8	3360	48	3298	193
4	1036	7200	44.9	9.2	41.3	4.6	3233	662	2974	331
	1100	10600	52.9	4.2	38.1	4.8	5607	445	4039	509
	1089	13100	60.3	3.3	27.3	9.1	7899	432	3576	1192
	1157	9700	46.5	2.9	44.4	6.2	4511	281	4307	601
	1492	9200	44.3	4.3	45.4	6.0	4076	396	4177	552
	1686	7000	46.8	6.6	40.5	6.1	3276	462	2835	427
							39085	3984	33049	4481
		8956	Average.				4343	443	3672	498
	1287	7600	54.0	3.3	36.1	6.6	4104	251	2744	502
	1432	7100	48.3	2.0	45.5	4.2	3429	142	3231	298
5	1686	7000	46.8	6.6	40.5	6.1	3276	462	2835	427
	1740	18000	43.3	6.4	46.3	4.0	7794	1152	8334	720
	1741	6100	44.7	7.0	42.4	5.9	2727	427	2586	360
	1854	8200	63.1	5.1	23.5	8.3	5174	418	1927	681
	2264	12500	59.2	2.3	32.1	6.4	7400	288	4013	800
							33904	3140	25670	3788
		9500	Average.				4843	449	3667	541
	1031	9900	53.1	5.9	35.7	5.3	5257	584	3534	525
	1316	9500	50.0	6.5	38.6	4.9	4750	618	3667	466
	1117	6100	40.3	12.6	40.1	7.0	2458	769	2446	427
6	1133	12800	52.1	5.1	33.1	9.7	6669	653	4237	1242
	1138	7500	51.8	1.2	38.2	8.8	3885	90	2865	660
	1229	5400	60.1	4.8	26.2	8.9	3245	259	1415	481
	1759	10000	50.8	12.6	31.6	5.0	5080	1260	3160	500
							31344	4233	21324	4301
		8744	Average.				4478	606	3046	614
	1396	11100	50.4	3.0	39.3	7.3	5594	333	4362	810
	1086	8700	45.7	2.7	42.7	8.9	3976	235	3715	774
	1202	9300	40.0	11.1	38.3	10.6	3720	1032	3562	986
	872	7900	51.4	4.1	27.4	7.1	4061	324	2955	561
7	1857	6500	36.1	7.6	53.8	2.5	2347	494	3497	163
	1850	12500	35.6	10.6	44.5	9.3	4450	1325	5563	1163
	2293	9200	48.2	2.0	40.3	9.5	4434	184	3708	874
							28582	3927	27362	5331
		9315	Average.				4083	561	3909	762
							43.83	6.02	41.97	8.18

## THE BLOOD PICTURE OF CHILDREN WITH ADENOID GROWTHS—Continued.

Age	Case-book No.	Total Number of leucocytes	PERCENTAGE				PARTIAL VALUES			
			Polynuclears	Eosinophiles	Lymphocytes	Monocytes	Polynuclears	Eosinophiles	Lymphocytes	Monocytes
8	962	5200	45.0	12.4	46.0	6.6	2340	125	2392	343
	962	7600	52.3	1.3	41.8	4.6	3975	99	3177	350
	1024	11800	58.3	3.3	31.8	6.6	6879	389	3752	779
	1206	4500	49.2	3.7	37.3	9.8	2214	167	1679	441
	1269	6500	48.8	11.4	34.6	5.2	3172	741	2249	338
	1272	7200	42.2	3.0	41.9	12.9	3038	216	3017	929
	1623	13200	45.2	3.1	46.1	5.6	5966	409	6085	739
	1616	14000	43.2	11.4	41.0	4.4	6048	1596	5740	616
	1647	11900	56.2	1.6	27.2	15.0	6688	190	3237	1785
							40320	3932	31328	6320
							4480	437	3481	702
		9100 Average.					49.23	4.8	38.25	7.72
9	856	5900	47.8	2.7	41.1	8.4	2820	159	2425	496
	966	8300	46.7	3.5	38.3	11.5	3876	291	3179	955
	1074	4300	60.9	3.1	32.6	3.4	2619	133	1402	146
	944	4600	50.1	2.8	40.5	6.6	2305	129	1863	304
	1185	6800	58.3	5.2	28.2	8.3	3964	354	1918	564
	1671	9500	52.8	3.4	33.1	10.7	5016	323	3145	1017
	1668	7400	38.5	4.3	48.0	9.2	2849	318	3552	681
	1718	8700	34.9	8.8	53.7	2.6	3036	766	4672	226
	1826	8700	60.1	4.2	31.6	4.1	5229	365	2749	357
							31714	2838	24905	4746
							3524	315	2767	527
		7123 Average.					49.40	4.42	38.79	7.39
10	1096	6100	53.7		37.6	8.7	3276	0	2294	531
	1132	7300	41.4	8.2	40.9	9.5	3022	599	2986	694
	1232	7700	62.7	1.6	31.1	4.6	4828	123	2395	354
	1496	9900	65.2	1.8	25.9	7.1	6455	178	2564	703
	1760	9500	70.4	1.8	22.4	5.4	6688	171	2128	513
	1833	11700	51.1	1.9	43.7	3.3	5979	222	5113	386
	971	9600	47.5	3.5	43.7	5.3	4560	336	4195	509
							34808	1629	21675	3690
							4973	233	3096	527
		8829 Average.					56.33	2.64	35.06	5.97
11	1108	7000	63.6	5.3	26.6	4.5	4452	371	1862	315
	1349	7500	58.3	4.9	30.6	6.2	4373	368	2295	465
	1099	4600	63.5	3.0	26.7	6.8	2921	138	1228	313
	1183	8400	49.6	9.9	29.9	10.6	4166	832	2512	890
	1232	6100	68.5	2.1	23.8	5.6	4179	128	1452	342
	2249	8400	52.4	4.0	38.6	5.0	4402	336	3242	420
	2235	7900	58.8	3.0	27.1	11.1	4645	237	2141	877
							29138	2410	14732	3622
							4163	344	2105	518
		7130 Average.					58.39	4.82	29.52	7.27

THE BLOOD PICTURE OF CHILDREN WITH ADENOID  
GROWTHS—Continued.

Age	Case-book No.	Total Number of leucocytes	PERCENTAGE				PARTIAL VALUES			
			Polynuclears	Eosinophiles	Lymphocytes	Monocytes	Polynuclears	Eosinophiles	Lymphocytes	Monocytes
12	1394	9200	54.0	3.1	38.0	4.9	4968	285	3496	451
	1135	9000	71.0	2.0	22.0	5.0	6390	180	1980	450
	1207	7700	59.7	1.1	32.6	6.6	4597	85	2510	508
	1115	6400	53.5	6.7	33.7	6.1	3424	429	2157	390
	1676	5600	52.9	3.3	40.7	3.1	2962	185	2279	174
	1780	6900	54.5	3.2	37.4	4.9	3761	221	2581	338
	1815	6700	58.5	3.2	35.1	3.2	3920	214	2352	214
						30022	1599	17355	2525	
						4289	228	2479	361	
	7357	Average.					58.30	3.09	33.70	4.91
13	6700	52.9	5.7	32.1	9.3	3544	382	2151	623	
	4800	46.8	1.1	38.7	13.4	2246	53	1858	642	
	10100	56.3	1.6	33.9	8.2	5686	162	3424	828	
	6900	56.5	3.2	39.4	0.9	3899	221	2719	62	
	7300	59.1	5.2	29.6	6.1	4314	380	2161	445	
						19689	1198	12313	2601	
						3938	238	2463	520	
	7159	Average.					55.01	3.32	34.40	7.27
14	10400	64.8	0.3	30.1	4.8	6739	31	3130	499	
	9000	55.1	2.7	35.9	6.3	4959	243	3231	567	
	7500	42.5	3.5	48.8	5.2	3188	263	3660	390	
	11400	66.8	1.6	26.0	5.6	7615	182	2964	638	
	8400	60.6	0.5	29.4	9.5	5090	42	2470	798	
						27591	761	15455	2892	
						5518	152	3091	578	
	9339	Average.					59.09	1.63	33.10	6.18

## THE POLYNUCLEARS IN NORMAL AND ADENOID CHILDREN.

Normal Children.				
	3-4-5 Years	6-7-8 Years	9-10-11 Years	12-13-14 Years
	42.40	55.61	52.73	48.40
	47.89	49.66	48.59	50.81
	48.05	48.49	55.20	56.69
	138.34	153.76	156.52	155.90
Average	46.12	51.25	52.17	51.97
Adenoid Children.				
	3-4-5 Years	6-7-8 Years	9-10-11 Years	12-13-14 Years
	41.79	51.21	49.40	58.30
	48.48	43.83	56.33	55.01
	50.98	49.23	58.39	59.09
	141.25	144.27	164.12	172.40
Average	47.08	48.09	54.71	57.47
Age	Normal	Adenoid	Difference between Normal and Adenoid	
3- 4- 5	46.12	47.08	— 0.96	
6- 7- 8	51.25	48.09	+ 3.16	
9-10-11	52.17	54.71	— 2.54	
12-13-14	51.97	57.47	— 5.50	
	201.51	207.35	— 5.84	
Average	50.38	51.84	— 1.46	

## THE EOSINOPHILES IN NORMAL AND ADENOID CHILDREN.

Normal Children.				
	3-4-5 Years	6-7-8 Years	9-10-11 Years	12-13-14 Years
	4.09	3.48	3.69	4.83
	5.32	6.01	2.48	2.93
	4.99	3.85	4.05	2.96
	14.40	13.34	10.22	10.72
Average	4.80	4.45	3.41	3.57
Adenoid Children.				
	3-4-5 Years	6-7-8 Years	9-10-11 Years	12-13-14 Years
	4.86	6.93	4.42	3.09
	4.95	6.02	2.64	3.32
	4.73	4.80	4.82	1.63
	14.54	17.75	11.88	8.04
Average	4.85	5.92	3.96	2.68
Age	Normal	Adenoid	Difference between Normal and Adenoid	
3- 4- 5	4.80	4.85	+ 0.05	
6- 7- 8	4.45	5.92	+ 1.47	
9-10-11	3.41	3.96	+ 0.55	
12-13-14	3.57	2.68	+ 0.89	
	16.23	17.41		
Average	4.06	4.35	+ 0.29	

## THE LYMPHOCYTES IN NORMAL AND ADENOID CHILDREN.

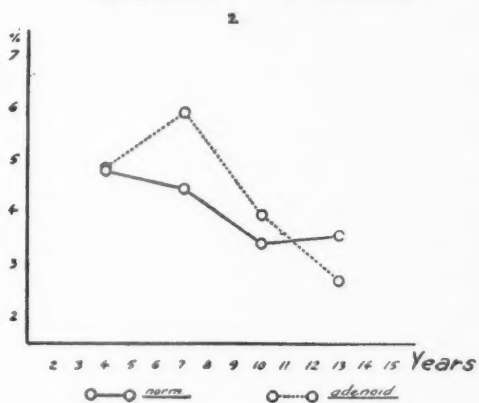
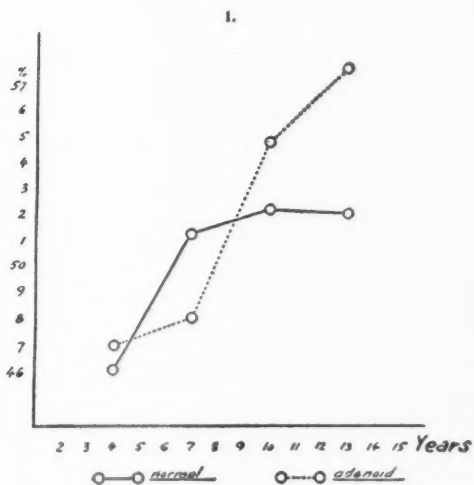
Normal Children.				
	3-4-5 Years	6-7-8 Years	9-10-11 Years	12-13-14 Years
	47.32	36.54	36.79	40.50
	41.47	38.09	44.55	40.57
	41.67	43.07	35.07	34.53
Average	130.46	117.70	116.41	115.40
	43.49	39.23	38.80	38.47
Adenoid Children.				
	3-4-5 Years	6-7-8 Years	9-10-11 Years	12-13-14 Years
	45.23	34.84	38.79	33.70
	41.00	41.97	35.06	34.40
	38.60	38.25	29.52	33.10
Average	124.83	115.06	103.37	101.20
	41.61	38.35	34.46	33.73
Age	Normal	Adenoid	Difference between Normal and Adenoid	
3- 4- 5	43.49	41.61	+ 1.88	
6- 7- 8	39.23	38.35	+ 0.88	
9-10-11	38.80	34.46	+ 4.34	
12-13-14	38.47	33.73	+ 4.74	
Average	159.99	148.15	+11.84	
	40.00	37.04	+ 2.96	

## THE MONOCYTES IN NORMAL AND ADENOID CHILDREN.

Normal Children.				
	3-4-5 Years	6-7-8 Years	9-10-11 Years	12-13-14 Years
	6.19	4.37	6.79	6.47
	5.32	6.24	4.38	5.69
	5.29	4.59	5.68	5.82
Average	16.80	15.20	16.85	17.98
	5.60	5.07	5.62	5.99
Adenoid Children.				
	3-4-5 Years	6-7-8 Years	9-10-11 Years	12-13-14 Years
	8.12	7.02	7.39	4.91
	5.57	8.18	5.97	7.27
	5.69	7.72	7.27	6.18
Average	19.38	22.92	20.63	18.36
	6.46	7.64	6.88	6.12
Age	Normal	Adenoid	Difference between Normal and Adenoid	
3- 4- 5	5.60	6.46	- 0.86	
6- 7- 8	5.07	7.64	- 2.57	
9-10-11	5.62	6.88	- 1.26	
12-13-14	5.99	6.12	- 0.13	
Average	22.28	27.10	- 4.82	

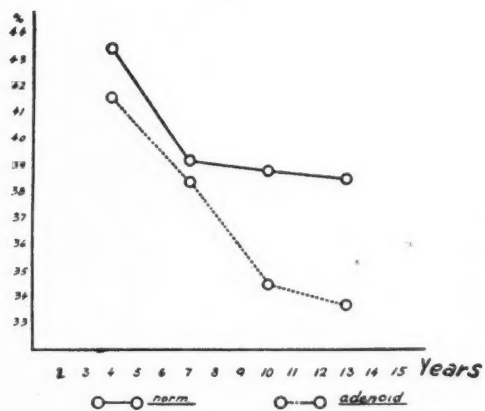


1. Polynuclear. 2. Eosinophile.

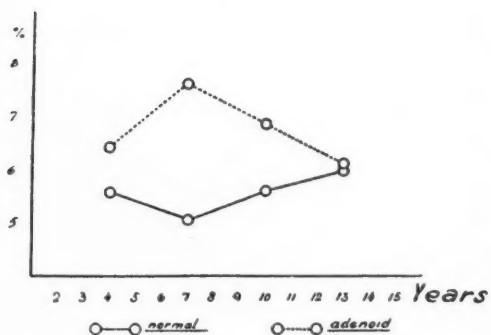


## 1. Lymphocytes. 2. Monocytes.

1.



2.



## SUMMARY OF CHAPTERS IV AND V.

1. In the material before us the gradual decrease of lymphocytes throughout childhood reported by previous authors has been substantiated. However, very great variations in the lymphocyte count in different children within the same ages seem to occur. By taking the average values in groups covering a period of three years, the individual variations seem to be eliminated, and a decrease, which has its maximum at the age of eight, is found. (See curve 1.)

2. A corresponding increase in the leucocyte count is seen. This increase is also on its maximum about the eighth year. (See curve 1.)

3. The eosinophile cells diminish in number throughout childhood. By taking periods of three years, a rather even decrease is found until the age of ten. (See curve 2.)

4. The figures for the monocytes are rather constant throughout the years from three to fourteen.

5. In children with adenoid growths the proportion of lymphocytes in the blood is, on an average, lower than in normal children; particularly after the seventh year, by taking the average periods covering three years, the lymphocyte count of adenoid children is so considerably lower than that of normal children that, evidently, this cannot be due to casualties.

6. As a higher total count (8,435 on an average, as against 7,674 in normals) and a higher proportion of polynuclear neutrophils are found in adenoid children, these factors have been taken to indicate an infectious state in such children.

7. In children with adenoid growths an increase in the proportion of eosinophile cells in certain age groups about the seventh year was demonstrated. This factor could not be referred to dermal affections in the children examined, nor, as far as could be ascertained, to helminthiasis.

## CHAPTER VI.

## ON LEUCOCYTOSIS IN INFECTIOUS DISEASES.

The term leucocytosis was originally defined by Virchow. He understood by leucocytosis a more or less transient increase in the number of white cells in the circulation, in con-

tradistinction to the persistent increase in leucemia. The introduction of Ehrlich's staining technic led to a much sharper distinction between the nosologic terms leucocytosis and leucemia, as the increase in the blood cells found in the latter disease was due to quite other forms of cells than those of the five main groups in Ehrlich's staining method—i. e., (1) neutrophiles, (2) eosinophiles, (3) basophiles (mast cells), polynuclears, (4) lymphocytes, and (5) monocytes. Since Ehrlich's time the views as to the origin of these five kinds of cells have been much divided. However, the dualistic theory still prevails, in spite of many new opinions, based on histologic investigations as to the prephases of the different kinds of cells, in holding the bone marrow as the place of formation for the polynuclears, and the lymphatic apparatus and the spleen for the lymphocytes. Without further discussion of the numerous investigations on the prephase of the different forms of cells, we shall briefly mention that, pursuant to the dualistic theory, a hyperfunction of the bone marrow is supposed to take place in polynuclear leucocytosis, while lymphocytosis is referred to as hyperfunction of the lymphatic apparatus. As far as the relative lymphocytosis is concerned, where it is only a question of a shifting in the blood count in the lymphocytic direction without any true increase of the lymphocytes, a hypofunction of the bone marrow is supposed to be the etiologic factor. For example, in the postinfectious relative lymphocytosis a hypoleucocytosis is found at the same time. The fundamental idea in the explanation of infection leucocytosis is, as far as most authors are concerned, chemotactic influences of the bacterial products derived from the affected tissue, on the leucocyte forming tissue, and, according to the nature of the infection, characteristic increases of one single form of cells is found to be specific for the infection in question.<sup>8</sup> In by far the majority of infectious diseases it holds good that the leucocytosis is due to an increase of the polynuclear neutrophile leucocytes. Only a few diseases form an exception, viz., typhus abdominalis, morbilli, robeulæ, malaria, apart from the febrile stages; in chickenpox a decrease of the neutrophiles is often found, as is also the case with certain tropical infectious diseases; it would, however, carry us too far afield to enter into this question in detail.

Infection leucocytosis played a rather important part as a diagnostic help a few years ago, even in surgery; thus, Sonnenburg tried to make his indications as to operation for appendicitis according to increases in the number of leucocytes. Arneth's kernel fragment count was used to a great extent, the main idea of which was that an increase of the youngest polynuclears with few kernel fragments meant an increasing infection. The practical application of the method was defeated by the fact that the blood picture was only an expression of the immediate resorption of reactive inflammatory substances. Thus a malignant appendicitis, where the resorption was diminished by an immediate blocking up of a place in the peritoneum fatal to the further course of the disease, may produce a blood picture indicative of a favorable course of the disease; and, moreover, it was not possible, in spite of Arneth's theory, fully to judge of the relatively slight leucocytosis or leucopenia found in by far the severest cases of infection.

In most cases of leucocytosis attending infection it holds good that it ceases, or, at any rate highly approaches the limit of the normal number below 10,000 simultaneously with deferescence. This is especially true of the various infectious diseases which are accompanied by leucocytosis, to a smaller degree of the suppurative processes (e. g., abscesses), wherever such may be localized in the organism—e. g., in the subcutaneous tissue, or in the liver, the lungs, the brain, glands, etc. In such cases a leucocytosis is often found to persist for a long period independent of the temperature, which only now and then by an insignificant rise discloses some occult focus of infection; in such cases a leucocytosis may afford more distinct evidence of an existing infection than the relations of the pulse or temperature. An exception to the rule that leucocytosis ceases after the acute stage of an infectious disease is afforded by scarlet fever.

## CHAPTER VII.

### SCARLATINA.

The rather numerous works on leucocytosis in scarlet fever (Türk, Kotschekow, Tilleston and Locke, Bennecke, Escherich and Schick, Reckzeh, Rieder, Felsenthal), seem to agree in

stating that this is the only infectious disease within epidemiology in which a leucocytosis persists after the acute stage, even when it is not a question of complications. Another rule, to which scarlatina forms an exception, is that the eosinophiles disappear or diminish to a fraction of a per cent in the acute febrile infectious stage. Most of these authors agree that in scarlatina there is frequently an increase of the eosinophiles from the exanthematous stage, which most usually coincides with a highly febrile state. The leucocytosis may persist for weeks, as may also the eosinophilia, but, as a rule, the latter vanishes in the course of ten days to a fortnight.

In Denmark, Skat Baastrup has tried to account for the difference between the blood picture of scarlatina and that of simple angina in order to find an aid to differential diagnosis. As his work forms the starting point for a section of the present study, the brief case histories will be given below of the first five angina cases and first five scarlatina cases out of the author's fifteen and ten patients, respectively, who were examined from day to day until the leucocytosis ceased. Furthermore, I give a brief summary of my investigations which, in addition to the above mentioned 15 and 10 patients, comprised 87 scarlatina patients, divided into groups from the second to the thirty-fifth day of the disease. All the angina patients had rather severe changes in the throat with marked swelling, redness, membranous exudate on the tonsils, and also fever, when admitted to the hospital. Thus, in six cases, the temperature was about 40° C., in five it was between 39° and 40° C., and in the remainder of the cases between 38° and 39° C. In the majority of cases the temperature became normal after the fifth day of disease. It appeared that the variations in the numbers of leucocytes ran fairly parallel with the oscillations in the temperature. Heavy falls in the temperature were accompanied by a very marked decrease in the number of leucocytes, while a more gradual decline in the temperature was accompanied by a corresponding slow decrease of the leucocytosis. Simultaneously with the temperature becoming normal the leucocytosis ceased. Previous authors, especially Bennecke, state that in angina tonsillaris the leucocytosis has, as a rule, disappeared by the third day of the disease, while several of Baastrup's patients had leucocytosis all through the

first week. The eosinophiles had either completely disappeared or only a fraction of a per cent was found in the febrile stage, after which an increase took place, which, however, in a single case was seen to begin while there was still a slight elevation of temperature (eighth day,  $6^{\circ}$  C., 5.6 per cent of eosinophiles). In a few cases a slight increase of the temperature was found on the third and fifth day,  $37, 7^{\circ}$  C., coinciding with a percentage of two to three of eosinophile cells. Apart from this increase just at the end of the febrile stage, no values were found in the postinfectious stage which rightly might be taken to indicate a postinfectious eosinophilia; as a rule, however, the patients were not examined beyond the first week.

As to the cases of scarlatina, which were all marked, or, at any rate, moderately severe cases, it is to be observed that in none of the ten cases which were thoroughly examined did the leucocytosis last for less than 20 days. In six cases the leucocytosis lasted even for from 30 to 43 days. In the 87 patients who were examined in groups on different days of the disease, leucocytosis was found in 100 per cent of the cases up to the seventh day of the disease (54 cases in all). No patients were examined between the seventh and the fourteenth day. On the fourteenth day, seven out of the eight patients examined had a leucocytosis of from 10,100 to 18,000, which was markedly exceeded only by those found in six patients out of the twenty-seven examined during the first four days of the disease, viz.: from 19,000 to 25,000. On the 21st, the 28th and the 38th day of the disease, 8, 8, and 9 patients, respectively, were examined, of whom 7, 5, and 4, respectively, had a leucocytosis, which on the 21st day of the disease, as far as two cases were concerned, showed counts of about 16,000 to 17,000. In the remaining, the counts ranged between 10,000 and 14,000, while it rose from 14,000 to 15,000 in two cases on the 28th day of the disease, and above 14,000 in one case on the 35th day of the disease.

The count of the eosinophiles in the ten patients will be noted in the accompanying table. The most interesting feature is that no ratio was found between the degree of the exanthema and the extent of the eosinophilia, just as the latter often increased after the cessation of the exanthema, while it was

never found before the exanthematous stage. The eosinophilia disappeared between the 10th and the 14th day in three cases. In six cases it disappeared between the 14th and the 28th day, and in one case it was found to persist on the 43rd day of the disease. On the eighty-seven patients, of whom nine were examined on the 5th day of the disease, eight had eosinophilia; on the 6th day, seven out of ten, and, on the 7th day, six out of eight showed an excess of eosinophiles; only in about one-third of the cases in these groups did the eosinophile cells reach above 10 per cent. In 78 per cent of the 54 patients examined during the first week eosinophilia was found, while leucocytosis was found in 100 per cent, as already stated. On the 14th day of the disease, five out of eight cases; on the 21st day, four out of eight, and on the 28th day, three out of eight patients had an excess of eosinophiles, which, in these three groups, taken all together, amounted to 10 per cent in four cases only. In the 87 patients the percentage of eosinophiles was never found to go below 2.1.

Summarizing the findings regarding eosinophilia, it may be said that the phenomenon was present in somewhat more than half of the cases at the end of the second week, in nearly half of the cases at the end of the third week, and, in somewhat above one-third of the cases at the end of the fourth week.

In regard to the diagnostic value of the findings, the following conclusions may be drawn:

1. In the febrile period leucocytosis in connection with eosinophilia, or merely a normal number of eosinophiles, will almost with certainty indicate scarlatina and exclude simple angina. Leucocytosis combined with complete absence of or a very small number (less than 1 per cent) of eosinophiles will exclude scarlatina and indicate a simple angina.

2. Subsequent to the febrile period, a leucocytosis combined with eosinophilia will exclude that the case is one of simple angina and will suggest scarlatina as the more probable diagnosis. Leucocytosis alone, or eosinophilia alone, will not to the same degree be of differential diagnostic value, but, on the other hand, either of these isolated phenomena may be indicative of scarlatina.



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TABLE OF BLOOD STUDIES IN CASES OF ANGINA (BAASTRUP).

No. 1. Day of Disease Age 21—Sex q.	1	2	3	5	8	10
	Swelling and redness of fauces. Whitish spotty membrane on tonsils.	Membrane somewhat increased.	Scattered small spots on tonsils.	Membrane, some swelling and redness of tonsils.	Slight swelling and redness only.	Slight swelling redness.
Clinical Remarks:	T. 40° C.	T. 39.9	T. 38.1	T. 37.1	T. 37.1	T. 37.2
No. of leucocytes.....	16500	17000	12000	10100	8600	8200
Neutrophile						
polynuclears .....	83.5	85.2	80.0	75.0	69.5	65.0
Eosinophiles .....	0.0	0.0	0.1	0.5	3.2	5.1
Basophiles .....	0.0	0.0	0.0	0.0	0.1	0.2
Lymphocytes .....	14.5	6.8	10.9	15.6	22.2	25.3
Monocytes .....	2.0	8.0	9.0	8.9	5.0	4.4
No. 2. Day of Disease Age 20—Sex q.	1	2	3	5	7	9
	Swelling and redness of fauces; cohesive membrane on left tonsil of moderate extension.	Membrane decreased.	Small spots on left tonsil.	A single little spot. Vigorous redness.	Membrane. Some redness.	Indisignificant redness.
Clinical Remarks:	T. 40.5	T. 39.8	T. 38.4	T. 37.7	T. 37.3	T. 37.1
Leucocytes .....	18200	14000	11100	9900	8100	8500
Neutrophile						
polynuclears .....	80.2	78.1	70.0	65.2	66.0	68.2
Eosinophiles .....	0.0	0.0	0.5	2.5	3.1	3.0
Basophiles .....	0.0	0.0	0.0	0.1	0.0	0.2
Lymphocytes .....	13.8	14.3	20.5	28.1	27.9	24.4
Monocytes .....	6.0	7.6	9.0	4.1	3.0	5.2
No. 3. Day of Disease Age 20—Sex q.	1	2	3	4	5	8
	Marked swelling and redness of fauces. Some lacunar plugs.	Fauces unchanged.	Swelling and redness of fauces; plugs.		Slight redness, some swelling.	No redness, some swelling.
Clinical Remarks:	T. 39.8	T. 38.5	T. 37.8	T. 37.4	T. 37.2	T. 37.3
Leucocytes .....	14500	12000	10200	7900	8000	8400
Neutrophile						
polynuclears .....	88.0	80.1	75.2	69.0	67.2	70.0
Eosinophiles .....	0.0	0.0	0.0	0.8	4.6	4.2
Basophiles .....	0.0	0.0	0.0	0.2	0.0	0.1
Lymphocytes .....	10.5	15.8	18.4	25.1	26.2	22.1
Monocytes .....	1.5	4.1	6.4	4.9	2.0	3.6

TABLE OF BLOOD STUDIES IN CASES OF ANGINA (BAASTRUP)—Cont'd

No. 4. Day of Disease Age 20—Sex q.	1	2	3	4	7
	Swelling and redness of fauces. No membrane.	Unchanged.	Less swelling and redness.	Slight swelling and redness.	Fauces normal.
Clinical Remarks:	T. 39.1	T. 38.9	T. 37.9	T. 37.2	T. 37.3
Leucocytes .....	13000	14000	11800	10000	7200
Neutrophile polynuclears .....	79.0	78.5	70.6	67.2	66.3
Eosinophiles .....	0.1	0.3	0.9	4.2	5.1
Basophiles .....	0.0	0.0	0.1	0.0	0.1
Lymphocytes .....	11.2	14.1	19.8	24.6	23.3
Monocytes .....	9.7	7.1	8.6	4.0	5.2

No. 5. Day of Disease Age 17—Sex q.	1	2	3	5	8	10
	Marked swelling and redness of fauces. Membrane on tonsils of moderate extension. Jugular glands swollen, the size of nuts.	Ulceration on right tonsil. the size of a lentil.	Membrane decreasing.	Isolated spots on tonsils. Ulcerations clearing up.	No membrane. Some swelling of fauces.	Still some swelling of tonsils.
Clinical Remarks:	T. 39.5	T. 39.2	T. 38.9	T. 37.9	T. 37.2	T. 37.1
Leucocytes .....	15300	14900	14700	11300	9100	9200
Neutrophile polynuclears .....	87.0	82.2	80.0	73.2	65.8	68.2
Eosinophiles .....	0.0	0.3	0.1	0.8	3.7	2.2
Rasophiles .....	0.0	0.0	0.0	0.0	0.0	0.0
Lymphocytes .....	8.6	12.5	16.8	22.0	25.3	25.5
Monocytes .....	4.4	5.0	3.1	4.0	5.2	4.1

Day of Disease	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
Scarlatina			1.4 19	2.1 19		4.2 19			6.4 21			7.1 16				1.8 15	
Angina	0 16	0 17	0.1 12		0.5 10			3.2 8		5.1 8							
Scarlatina	0.6 16	5 11	5.6 14	8.9 8	9.8 13	9 12	6.6 12	5.8 11			4.8 11					3.8 10	
Angina	0 18	0 14	0.5 11		2.5 9		3.1 8		3.0 8								
Scarlatina	2.2 19	4.8 17	5.2 19	5.7 16	6.4 14		5.4 14			4.3 17				(25)			15
Angina	0 14	0 12	0 10	0.8 7	4.6 8			4.2 8									
Scarlatina			9.2 13	11.8 11	12.3 14			10.8 10		8.1 11				7.1 15			
Angina	0.1 13	0.3 14	0.9 11	4.2 10			5.1 7										
Scarlatina	13 24	15.8 24	14.9 21	14.7 17			11.5 16		9.9 12					8.6 13			
Angina	0 15	0.3 14	0.1 14		0.8 11			3.7 9		2.3 9							
Scarlatina	5.5 14	6.5 16	8.5 15	9.8 13	9.1 10		8.1 12			7.6 11						5.9 10	
Angina	0.2 12	0.3 11	2.1 9	6.1 7	3.2 8												
Scarlatina	3.5 25	11.8 23	10.8 23	14 19	13.8 17	12.1 18		10.2 22					9.5 10				
Angina	0 17	0 16	0 11	0.7 11		5.6 8				3.3 9							
Scarlatina	17.5 12		20 11	17.5 14	13.5 15	11.5 16	9.5 15			7.5 12						6.2 10	
Angina	0 19	0.3 14	0.8 11		1.8 8					3.3 9							
Scarlatina	1.2 21	2.3 25	14.1 26	15.8 21	17.5 18	16.2 19	13.2 14	11.1 16	10.6 15	11.5 15	9.6 13	8.7 13				6.3 15	
Angina	0.4 11	3.2 8		2.4 7													
Scarlatina	2.5 9	18.5 22	28.5 18		13.5 21			7.4 12			34.3 19		20.1 12				
Angina	0.1 14	0.1 12	3.2 10		4.2 8												

The larger numbers indicate the leucocyte count; the smaller, the eosinophiles.

From the above examinations it will appear that there is a marked difference between the blood picture of the scarlatinous angina and that of the ordinary angina tonsillaris as regards leucocytosis and the number of eosinophile cells. It is seen that scarlatina has a permanent leucocytosis, which persists after the cessation of the febrile stage, often for several weeks into the convalescence. The numbers of leucocytes range between 8,000 and 26,000 in the first sixteen days of disease. Leucocytosis was found in all ten patients. The course of this leucocytosis was not quite regular; although, on the whole, it was the rule that the number of leucocytes decreased from the highest during the first days to lower later on. A single exception of this rule was found: Case 8 showed lower count (12,000-11,000) on the third and on the fifth day of disease, while a higher count was found on the following four days (14,000, 15,000, 16,000, 15,000). In Case 3, a single isolated high count (25,000) was found on the fourteenth day of disease in connection with an increase of temperature ( $38.8^{\circ}$ ) owing to a parulis. Moreover, during the course of the leucocytosis, the leucocyte counts showed variations which were not quite insignificant. In Case 2 the count fell from 16,000 to 8,000 on the fourth day in order to increase again to 13,000, 12,000 and 11,000 on the following days; in Case 7 the count declined from 21,000 on the second day of disease to 18,000 on the seventh day, and rose again to 22,000 on the ninth day. In Case 10 the figure was 9,000 on the second day of disease, on the third, fourth and sixth day it was 22,000, 18,000 and 21,000, respectively, on the ninth day 12,000, on the 12th 19,000, and on the 14th, 12,000.

In contradistinction to these findings those found in ordinary angina showed a quite regular decline from the highest on the first and second day of disease to normal as early as on the third, fourth, fifth or sixth day.

The following examination of a total of 165 patients, admitted to the Blegdam Hospital under observation for scarlatina, were undertaken under uniform conditions. The samples of blood were taken in the middle of the day between 11 and 1 o'clock, several hours after a meal. The patients were all in bed, and the specimens were taken from the ear according to the routine already stated.

In this chapter where we are concerned with a possible leucocytosis (and eosinophilia) in mild cases of scarlatina, the histories have been classified according to the division at foot, all values being determined without any anticipative knowledge as to the issue of the experiments. In this connection it should be observed that during the work at the laboratory it was not known which specimens originated from patients suffering from scarlatina and which specimens were from other cases.

I. Undoubted scarlatina:

- (a) showing a leucocytosis with values exceeding the range of possible counting errors (above 12,000), 49 cases.
- (b) showing a leucocytosis, the values falling within the range of error (16,000-12,000), 11 cases.
- (c) no leucocytosis (below 10,000), 3 cases.

II. Not scarlatina:

- (a) no leucocytosis, 58 cases.
- (b) leucocytosis (a) pursuant to an infectious disease, 25 cases; (b) to noninfectious affections, 2 cases.

III. The diagnosis of scarlatina doubtful: Probable, possible, scarlatina not excluded, 14 cases.

IV. Cases which have leucocytosis without any traceable cause, 3 cases.

The mode of procedure was as follows: At certain intervals—usually every third day, at first somewhat less frequently—samples were taken according to the technic previously described, the cells being counted in the counting chamber and the percentage of eosinophiles in the preparation determined in proportion to the total number of cells. The percentages of the other kinds of cells were not determined, as the object of these investigations was to find out whether the total values for leucocytes showed any specific relations in mild cases of scarlatina as compared with the other cases which have been under observation for scarlatina. Thus, it is not a question of determining the entire blood count; for the same reason, the leucocytosis was not in all cases observed until it ceased. This, too, would have proved very difficult on account of the

removal of the patients to the scarlatina ward. Owing to lack of space, it has been necessary to make only quite brief abstracts from the casebooks, whereby it has been rendered somewhat difficult to the reader to judge of the diagnosis itself; it must, however, in these cases be remembered that the diagnosis, scarlatina or not, was made by the experienced physicians of the hospital.

If we consider the first group (Ia), the following table may afford a general survey:

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## Group 1A.

No. of leucocytes expressed per mille.						Corresponding contents of eosinophiles expressed per cent.					
14.5	13.3					5	6				
16.3	20.3	19.3	14.6	12.0		3	3	5	6	8	
11.7	10.8	11.3	10.5	17.2		6	5	3.6	4	2	
15.9	20.0	14.0	10.0	16.0	10.2 10.8	3.5	2	2.5	5	1	2 5
12.9	18.0					10	8				
10.9	15.9	18.0				11.6	16.4	9.5			
20.0	9.0	14.9	12.2	11.7	9.4	7	10	6	8	2	3
9.9	13.4	8.6				6	6	6			
15.0	15.2					7.5	9.5				
14.9	15.3	13.8	11.7	12.5	10.6 12.8	13	9	4	3	7	10 7
14.9	17.2	15.2				5	10	5			
10.1	12.0	12.3				2	5	4			
13.2	10.5					6	3				
10.5	13.2	10.4				4	3	1			
17.2	9.9					2	5				
14.0	11.7					1	3				
12.5	13.1					4	2				
11.3	12.3	12.9	13.3	13.2		0.5	2	3	0.5	3	
11.0	11.4	17.0	13.9			1	1	1.8	0.7		
10.3	12.0	12.2	16.5			7.2	9.1	8	11.1		
14.4	15.7	14.9	16.5			5.4	4	0.8	0.9		
22.9	22.0	12.4	12.2	10.2	12.4 13.2 16.2 14.3	1.7	0.25	0.7	1	0.6	1.5 1.9 2.6 1.6
8.4	8.8	11.3	12.2			1	1.6	6	6		
8.1	11.8	11.6	13.8			0.5	5	4	4		
8.3	13.9	12.2	13.5	11.8	10.2	13	6.9	4.1	10.7	11	4.3
12.9	12.1	10.2	9.8	9.9		5.9	6.5	6.3	8.5	7.6	
13.1	18.6	9.5	10.7			1.9	3.2	4	3.5		
7.6	8.2	9.6	19.8	13.4		2.8	4.9	4.8	4.8	5	
12.9	11.0	10.8	11.7			9	14.2	14	14.3		
14.5	14.2	12.4	9.1			0.5	1	3.8	1		
11.4	12.2	8.9	9.8			3	1.5	2.6	0.5		
14.8	13.2	18.4	11.5			1.5	2.1	2	3.1		
8.2	10.2	13.6	12.4			2.8	5.5	7.5	8		
13.2	12.2	15.6	10.1			2	1	1	3		
15.6	16.7					1.5	2				
9.8	12.4					3	4				
9.3	10.4	11.2	12.5			1	2	4	7		
11.6	12.4	14.6	10.2			11.5	3	9.1	5.7		
13.2	14.0	15.6	10.8			0	1.2	3.2	2.2		
13.8	16.4	12.8	9.9			3.7	3.7	2.4	1.8		
13.8	14.3	11.0	15.3			2.1	1.5	1.8	5.2		
12.8	11.3	11.0	11.9			3.3	7.4	4.4	6.5		
13.8	12.4	10.1	9.9			5.1	2	2.2	2.5		
12.8	17.6	16.6	15.6			10.3	1.5	5.2	7.9		
14.4	15.6	10.8	14.7			4.3	2	6.1	6.8		
9.6	13.2					6.1	2.1				
12.6	11.1	10.3	14.1			10.6	7.6	2	4.1		
14.8	13.4	14.6	14.4			1.3	4	3.2	12.2		
15.1	10.6	12.4	10.1			3.1	3.2	2.9	3.7		

## Group 1B.

12.0	11.8		
9.6	10.2	9.2	
10.2	11.2	10.9	
11.6	9.6	9.5	
10.2	11.0	8.4	
9.2	10.8	10.1	9.7
9.3	9.9	7.9	11.7
11.8	10.8		
9.6	10.3	11.2	
10.4	11.3	11.6	11.2
10.4	9.6	10.3	10.1

## Group 1C.

9.8	9.4
9.8	9.6
8.8	9.6

TABLE SHOWING BLOOD INVESTIGATIONS IN CASES OF SCARLATINA.

[illegible]





TABLE SHOWING BLOOD INVESTIGATIONS IN CASES OF SCARLATINA—Continued.

[illegible]

TABLE SHOWING BLOOD INVESTIGATIONS IN CASES OF SCARLATINA—Continued.

[illegible]

In the foregoing table, Ia, comprising 49 cases of undoubted scarlatina, it appears that in a total of 191 counts determined in the first weeks of the disease

11	were above	18,000
14	were between	16,000 and 18,000
36	were between	14,000 and 16,000
57	were between	12,000 and 14,000
48	were between	10,000 and 12,000
24	were between	8,000 and 10,000
1	was between	7,000 and 8,000

In 9 out of the 49 cases the first count was below 10,000, and the leucocytosis did not appear until at the following examination. Case 23 and Case 28 call for special mention; in the former, a moderate leucocytosis and eosinophilia did not appear until at the third and fourth examination (11,300 with 6 per cent of eosinophiles, and 12,200 with 6 per cent of eosinophiles). In the latter case, a leucocytosis did not appear until the fourth examination (19,800), while the first three counts did not indicate the blood picture characteristic of scarlatina, neither as far as the total number of leucocytes was concerned nor in the proportions of eosinophiles.

Furthermore, no definite law seemed to govern the degree of the leucocytosis at the various stages of the disease. The number of cases in which the leucocytosis becomes more marked at a later stage nearly equals that of the cases in which it is more marked at the beginning. It will also be seen from the table that the number of leucocytes may vary rather considerably from one examination to the other; in the majority of cases, however, it holds good that two successive counts are fairly close on one another.

In the 49 cases of scarlatina the leucocytosis was marked—i. e., when examining several times, since in a number of cases—as has been pointed out—it appeared only gradually; if it is desired among these 49 patients to try to find out whether there is any demonstrable difference between the degree of the leucocytosis in the more severe cases and that of the milder cases, one would expect to fail in such an examination. In the first place, all the 49 cases were patients admitted to hospital for observation with such mild symptoms that the diag-

nosis was doubtful on admittance; in the second place, these patients had in many cases been in bed at home for a shorter or a longer period during the acute stage, so that owing to deficient information at their admission to the hospital it was often difficult accurately to judge the degree of the disease. To this must be added that, in the cases of moderately severe cases of scarlatina already mentioned, no true harmony was found to exist between the degree of the leucocytosis and the degree of the disease. To illustrate this it may be mentioned that Cases 9, 11, 21, 32 and 34 did not seem more severe than Cases 8, 12, 14, 23, 43, although the cases in the first group had a higher number of leucocytes than those of the last group, viz.:

Case					Eosinophiles:				
	9—15.0	15.2			7.5	9.5			
11—14.9	17.2	15.2			5	10	5		Normal temperature; slight exanthema.
21—14.4	15.7	14.9	16.5		5.4	4	0.8	0.9	Normal temperature; slight exanthema.
32—14.8	13.2	18.4	11.5		1.5	2.1	2	3.1	No characteristic exanthema.
34—13.2	12.3	15.6	10.1		2	1	1	3	T. normal; no exanthema; moderate desquamation.
									Temperature normal.

On the contrary, the following cases, which, at any rate, seem to be at least as severe as the above, as they have both increase of temperature and exanthema, may be given:

Case					Eosinophiles:				
	8—9	9	13.4	8.6	6	6	6		
12—10.1	12.0	12.3			2	5	4		Marked exanthema and increase of temperature.
14—10.5	13.2	10.4			4	3	1		Exanthema, increase of temperature.
23—8.4	8.8	11.3	12.2		1	1.6	6	6	T. increased, exanthema.
43—13.8	12.4	10.1	9.9		5.1	2	2.2	2.5	T. increased. General scaling. Adm. on 2nd day of disease.
									Considerable increase of temperature, exanthema, characteristic scaling.

While the leucocytosis was rather marked in these 49 patients, it appears that the eosinophilia is a far less reliable phenomenon. Even in moderately severe cases of scarlatina the eosinophilia, as already mentioned, was the one of the two factors which disappeared first in the characteristic blood picture. While in all of Baastrup's 10 cases the leucocytosis persisted until the 20th day, and, in 6 cases it lasted even more than 30 days, the eosinophilia disappeared between the 10th

and the 11th day in 3 cases, between the 14th and the 28th day in 6 cases. Among the 87 patients (Baastrup), who were examined only on a single day, only 7 out of 10 had eosinophilia on the 6th day of the disease, only 5 out of 8 on the 14th day, and only 4 out of 8 on the 21st day. As already mentioned, no marked agreement between the degree of the eosinophilia and that of the exanthema or of the severity of the disease, was found. It is therefore very probable that the eosinophilia in these 49 patients is a highly doubtful symptom, and the uncertainty is increased by the fact that only part of the patients were examined in the first days of the disease.

The first sample of blood was taken from Nos. 5, 12, 22, 27, 30 and 35 on the 2nd day of disease.

On the 3rd day of the disease from Nos. 20, 23, 39, 40, 41, 42, 44, 47, 48.

On the 4th day of the disease from Nos. 2, 7, 9, 14, 16, 18, 26, 28, 29, 38 43 45, 49.

From Nos. 8, 19, 21, 24 on the 5th day of the disease;

From Nos. 6 and 32 on the 6th day of the disease;

From Nos. 31, 34, 37 on the 7th day of the disease;

From Nos. 11, 25 on the 8th day of the disease;

From No. 1 on the 9th day of the disease;

From Nos. 3, 17, 36 on the 10th day of the disease;

From No. 8 on the 11th day of the disease;

From No. 13 on the 12th day of the disease;

From No. 10 on the 14th day of the disease;

From No. 15 on the 16th day of the disease;

From Nos. 4 and 33 on the 23rd day of the disease.

When taking only the cases in which the first sample was taken in the period from the second to the eighth day of the disease, altogether 38 cases—as will appear from the above table—it will be seen that eosinophilia was found in 12 cases only, viz.: In Nos. 5, 6, 7, 9, 11, 21, 25, 26, 29, 38, 45 and 46, which means that in mild cases of scarlatina, eosinophilia is found in about one-third of the cases only, even if the samples were taken during the week in which the moderately severe cases show eosinophilia, at any rate, in the majority of cases, although not as an absolutely constant symptom.

In Group Ib, dealing with the cases of doubtless scarlatina which show a leucocytosis within "the neutral zone"—i. e.,

below 12,000, the first count in Case 1 was made on the 15th day, in Case 8 on the 14th day, while amnesic information as to Case 3 is wanting. In the remaining eight cases the first counts were undertaken within the first nine days of the disease; of these cases, half—i. e., Nos. 2, 4, 6 and 7—show an increase of the eosinophiles.

In Group Ic, neither leucocytosis nor eosinophilia was found. Only two counts have been made for each patient. It is therefore possible that repeated tests would have shown a leucocytosis at a later stage (confer instances in Group Ia). Cases 1 and 2 have values lying near the limit for leucocytosis (9,800, 9,600, 9,400). In Case 2 the first count was made on the 11th day; in Case 3 the count was made during the exanthematous stage, and it is even possible that the true scarlatina exanthema did not appear until the day after the count (see abstract from the casebook).

Ila, comprising fifty-eight cases which were not scarlatina and in which there was no leucocytosis.

No. of leucocytes (per mille.).				Corresponding contents of eosinophiles (per cent).			
1—9.8	9.4	8.1		3.7	2	3	
2—9.5	9.2			4	3.5		
3—12.0	9.8	8.1		3	4	4	
4—10.2	9.1	8.2		6	2	2	
5—9.2	10.9			8	4		
6—8.3	8.9	9.0	8.3	3.5	2	2	2
7—8.6	9.3			4	2		
8—8.3	9.8			2	3		
9—9.1	9.4			2	1		
10—8.4	10.0			3	6		
11—9.9	9.6	9.5	9.4	3	4	4	7
12—9.2	9.8			5	2		
13—9.7	8.7			9	8		
14—8.9	8.4			4	4.5		
15—9.8	10.9	9.6		5	7	3	
16—11.4	10.2			5	2		
17—9.7	9.6			0	2		
18—10.8	10.7			4	3		
19—10.4	9.8	10.4		0	1	1	
20—10.3	7.8			0.5	2		
21—9.4	8.9	8.0		2.5	2	2	
22—7.6	9.3			1.5	3		
23—9.1	8.3			1.5	2		
24—9.3	9.6			0.6	0.2		
25—9.8	10.2			3.5	5		
26—8.9	9.9			0.5	6		
27—10.2	10.3	8.8	7.6	3	1	2	2
28—10.1	10.3			2	3		
29—7.6	9.4	10.3	10.1	1	3	2	1
30—7.2	10.5	9.6	8.4	2	1.5	2.5	1
31—8.2	8.4			1	2.5		
32—8.5	9.2			3	4		
33—10.1	9.4			1	2.5		
34—8.9	10.2			2	2		
35—6.4	9.8			1	1		
36—11.2	11.5			0.5	5		

No. of leucocytes (per mille.).				Corresponding contents of eosinophiles (per cent).			
37—6.8	9.8			4	2		
38—9.9	7.8			2	6		
39—9.7	9.9	11.0	7.9	1.5	3	3	1.5
40—9.8	10.8			0	2		
41—11.0	11.6	10.2	10.2	4	2	4	5
42—11.8	8.2			1	3		
43—8.4	7.3			2	1.2		
44—8.4	9.5			0	2.1		
45—6.4	9.3	9.8	9.9	1	2	2	1
46—8.4	9.4			0.5	1		
47—8.3	9.1	7.9		3	5	2	
48—9.3	8.6			4.5	6.2		
49—6.4	7.9	10.3	9.6	1	0.3	2.6	1.9
50—11.4	9.8	8.5	7.6	1.6	0.5	3	2.1
51—7.4	10.3	10.2	9.2	2.5	2.8	3.2	5.4
52—8.1	9.6			3	2.5		
53—9.6	10.5			1	0.3		
54—10.5	11.4	11.8	11.2	3	4	2	1.5
55—9.2	9.9			2.5	1		
56—7.5	7.3			0	3.5		
57—11.0	11.6			4	6.5		
58—9.8	8.2			3.2	0.9		

## GROUP IIa.

The most frequent diagnosis in this group was angina tonsillaris.

In 1918, cases No. 67 and 79; 1919, cases No. 36, 112, 161, 149, 207, 218; in 1920, cases No. 72, 79, 98, 110, 114, 131, 132, 165, 303; diphtheria was found in 12 cases in 1920, viz., cases No. 497, 550, 594, 626, 689, 710, 734, 1051, 1088, 1503, 1712 and 1720.

A non-specific exanthema was found in 14 cases to be the cause of the admission to hospital, viz., in 1918, cases No. 57 and 105; in 1919, cases No. 46, 99, 108, 141, 208; in 1920, cases No. 42, 113, 119, 130, 140, 142 and 160.

Scaling was the cause of the admission in four cases, viz., in 1919, case No. 157; in 1920, cases No. 66, 77 and 124. The remainder of the cases were: measles (2 cases), chickenpox (1 case), German measles (1 case), urticaria (1 case), bronchitis (2 cases).

The table shows that the count of leucocytes were:

Above 6,000 4 times;  
 Above 7,000 14 times;  
 Above 8,000 33 times;  
 Above 9,000 57 times;  
 Above 10,000 27 times;  
 Above 11,000 14 times;  
 Above 12,000 1 time.



Thus the borderland counts within the range of error, between 10,000 and 12,000, as found in forty cases altogether, have been included, while 104 counts were below 10,000.

It will be noted how relatively few lower counts are found: thus, none as low as 4,000 or 5,000 were found. The explanation is doubtless that it is a question of patients admitted in the very transitional stage between an acute febrile disease and convalescence, that the stage of convalescence proper was not included, during which one would expect to find a hypoleucocytosis.

The eosinophiles were as follows:

0	per cent of eosinophiles found	4 times;
Between 0 and 1	per cent of eosinophiles found	10 times;
Between 1 and 2	per cent of eosinophiles found	28 times;
Between 2 and 3	per cent of eosinophiles found	45 times;
Between 3 and 4	per cent of eosinophiles found	26 times;
Between 4 and 5	per cent of eosinophiles found	17 times;
Between 5 and 6	per cent of eosinophiles found	8 times;
Between 6 and 7	per cent of eosinophiles found	5 times;
Between 7 and 8	per cent of eosinophiles found	2 times;
Between 8 and 9	per cent of eosinophiles found	2 times;
Between 9 and 10	per cent of eosinophiles found	1 time

i. e., 140 counts were below 6 per cent, while ten were above 6 per cent, but of these values only five were above 7 per cent. When looking at the latter five it appears that two of them are found in the same patient (No. 13), who had suffered from angina; here it might be justifiable to consider the eosinophilia as postinfectious. The same may be said of Class 5. Of the remaining two cases, No. 11 and 15, which showed above 7 per cent, it holds good that both these patients had a scarlatinoid desquamation, this desquamation being the only symptom, however, it was considered to be due to other causes. These two cases form a transition to Group III: the scarlatina diagnosis is doubtful, and apart from these two doubtful cases no eosinophilia is found in Group II beyond the above described postinfectious one.

We now turn to Group IIb: leucocytosis in cases which were not scarlatina. Leucocytosis caused by infection was found in

25 cases. The diagnoses were: Angina, cases No. 62/18, 27/20, 59/20, 81/20, 84/20, 105/20. Tonsillitis ulcerativa: cases No. 54/19 and 39/20. Diphtheria (febrilia), cases No. 73/18, 656/20. Bronchopneumonia, bronchitis, cases, year 1919, No. 69, 98, 128, 153; year 1920, No. 28, 70, 145, 161, 378 and No. 1/1921. Adenitis acuta, No. 54/19 (also tonsillitis ulcerativa), No. 16/20 and 549/20. Pyuria (febrilia), No. 70/19. Otitis media suppurativa acuta (nonperforated), No. 146/19. Febrilia, case No. 40/20.

In all cases the leucocytosis is in harmony with the infectious suffering. It will only be necessary to attach special remarks to one group—i. e., that of adenitis acuta, where leucocytosis was found several times without fever, but where the temperature curve nevertheless revealed the infection by a slight rise now and again (37.6 to 37.7). Tenderness of the swollen glands was observed as an indication of an inflammatory process. The clinical demonstration of an inflammatory focus is thought to be a satisfactory explanation of the leucocytosis in these cases. From these cases of adenitis acute with leucocytosis, although few in numbers, it may be inferred that there are cases too in which a leucocytosis is found without our being able to demonstrate an inflammatory focus. Such a focus may be present in glands which cannot be palpated—e. g., in mediastinum or the deep jugular glands. Such cases of leucocytosis of an unaccountable origin have been exceedingly few in this study (see Group IV).

Leucocytosis (and possibly eosinophilia) of noninfectious origin (IIb) was found in two cases only, viz., in No. 165/19 (dermatitis medicamentalis) and No. 85/20: toxic exanthema of unknown etiology, leaving a vigorous pigmentation.

Considering the number of eosinophiles, we see that a percentage of 7 was found in one patient only out of 25 in Group IIb. This patient had an acute adenitis. No explanation of the very considerable eosinophilia (8, 13,  $7\frac{1}{2}$  per cent) in this patient could be found. Besides this one, there was only one other patient with eosinophilia (case No. 165/19 in Group Ib). The patient had a mercury dermatitis. The explanation of the eosinophilia may be that the patient had got salvarsan.

## GROUP II-B-1

## LEUCOCYTOSIS IN CASES WHICH ARE NOT SCARLATINA.

Leucocyte count (per mille.).				Corresponding contents of eosinophiles (per cent).			
1—17.6	11.8			5	2.5		
2—12.7	11.9			6.5	4		
3—16.1	12.1	14.4		5.5	3	3	
4—10.8	13.0			1.5	2		
5—18.6	9.8	11.4		6	5	2	
6—11.5	15.3	12.4		6	5	5	
7—18.7	12.1	11.9		1	4	1	
8—16.5	11.2			7	3		
9—12.1	14.2			2	3		
10—5.3	8.8	12.9		0.9	0.3	0.6	
11—8.2	13.3	18.3		2	4	6	
12—14.2	12.3			0.5	1.5		
13—10.9	13.2			2	4		
14—12.8	8.6			1.5	4.1		
15—12.2	14.6	10.2	11.4	1	0.8	4	0.5
16—13.2	13.4			0.5	5		
17—13.6	8.9			0.3	3		
18—12.6	10.5			1	3		
19—13.7	14.3	11.9		2.5	0.5	0.5	
20—10.9	16.3			1.5	3		
21—13.4	13.5	6.4	9.6	3.5	0.6	1.1	1
22—6.0	13.5	15.2	18.0	1	0.2	0.9	0.3
23—10.9	11.6	12.4	10.4	8	13	7.1	4.1
24—14.3	9.7	9.2		0.5	0.5	3	
25—12.6	13.2			5.1	4		

## GROUP II-B-2

1—19.8	10.2			8	9		
2—13.2	11.2	13.2		3	2.5	1	

The third group comprising the doubtful cases of scarlatina is not the least interesting, as it appears that just in the cases where scarlatina was suspected, and where this diagnosis is probable, possible or not excluded, leucocytosis or eosinophilia, or both, were found. With only few exceptions (1) a moderate leucocytosis was found in mild cases of scarlatina, and (2) no leucocytosis was found in cases which were not scarlatina, unless there was another traceable infectious condition. Therefore, I think it justifiable—using items I and II as a basis—to maintain that, when facing a case of doubtful scarlatina where there is real ground for supposing this diagnosis the probable one, but where the clinical symptoms are not quite clear, the presenec of leucocytosis, possibly in connection with eosinophilia, will support this supposition. It is scarcely safe to draw more definite conclusions. To take a leucocytosis (and, possibly, an eosinophilia) as a decisive symptom would hardly be justified, considering how many different ailments may be the causes of a leucocytosis or an eosinophilia, and remembering that an exanthema alone, a scaling down, or a "strawberry

tongue" alone is not sufficient ground for making the diagnosis of scarlatina, which is just based on an interaction of various symptoms. However, in connection with the preceding examinations the leucocyte count seems to be of some diagnostic value in doubtful cases of scarlatina, provided we do not rest content with a single count but examine for a period at certain intervals. The importance of this examination for practical diagnosis is diminished by these facts, but, theoretically, there remains the interesting discovery that these clinically very vague symptoms may call forth observable changes in the blood picture.

Group III is made up of fourteen cases of uncertain scarlatina, namely:

Case-book		Leucocyte counts (per cent)					
No.							
1—	58/18	13.9	14.0	14.0	11.1		Probably scarlatina sine exanthemata.
2—	96/18	12.1	11.8				Probably scarlatina.
3—	2/19	11.6	13.6	11.2	10	10	Scarlatina (?)
4—	6/19	12.6	12.8	12.5			Suspected for scarlatina.
5—	89/19	13.9	21.1	12.3			Scarlatina (6).
6—	118/19	9.4	9.3				Possibly scarlatina.
7—	129/19	14.7	15.2	10.2			Probably scarlatina.
8—	138/19	13.7	12.5	10.2			Almost ascertained scarlatina.
9—	192/19	17.4	14.2				Probably scarlatina.
10—	52/20	13.4	10.4	10.5	8.4		Scarlatina not improbable.
11—	88/20	13.6	12.3	16.3			Suspected for scarlatina.
12—	100/20	12.3	16.3	10.4	9.8		Probably scarlatina sine exanthemata.
13—	111/20	11.7	14.2	12.1	13.5	10.1	Angina, suspected for scarlatina.
14—	1032/20	10.1	11.8	12.1			Almost ascertained scarlatina.

Here, leucocytosis was found in all cases except one (No. 6). In this case the values were quite near the limit for leucocytosis, and, as regards eosinophiles, a content of 7 per cent was found, which corresponds to the total number of leucocytes, 9,400.

#### IV. LEUCOCYTES WITHOUT ANY TRACEABLE CAUSE.

This group comprises three cases in all; these were the only cases in the present series where an existing leucocytosis could not be brought in harmony with the clinical findings.

The three cases are the following:

Case 1.—No. 18/20, E. V. J., male, 24 years old. January 22—January 31. Patient had been on duty in scarlatina ward. Taken ill yesterday evening; lacunar membranes were observed on admission. Tongue with slightly swollen papillæ; during whole stay she had pains at swallowing, as also swelling and redness of tonsils. Temperature, from January 22 to

24, 37.8 to 37.7°. January 23, 14,500—4.1; January 27, 16,000—1.09; January 30, 12,700—4.3.

Case 2.—No. 19/20, H. H., female, 12 years old. January 26—February 1. Ill three days previously with angina. Exanthema is said to have been observed before admission on crura and a little on arms, not on trunk. Swollen papillæ on tongue, which is otherwise coated except on tip, where it is nude. January 31, tongue cleared, but not nude. No fever. January 27, 10,400—0.7; January 30, 15,600—2.6.

Case 3.—No. 171/20, M. L., male, 29 years old. December 10-20. Taken ill three days previously with fever. Exanthema observed day before admission. Tongue not scarlatinoid. On admission a typical universal exanthema is seen, and the patient is admitted to scarlatina ward, but is moved at night to observation ward. December 11, tongue not scarlatinoid. Exanthema on chest a little too large spotted to resemble scarlatina rash, but on abdomen, back and right arm it quite resembles scarlatina. December 12, exanthema faded, tongue normal. December 11, 11,600—12.3; December 15, 12,400—6.9; December 18, 8,400—3.2.

The first patient is of special interest, because swelling and redness of the tonsils are observed throughout her stay at the hospital, and it would, therefore, perhaps be thought reasonable to transfer this case to Chapter IX as a case of protracted angina with tonsillitis and leucocytosis; but as no attention has been bestowed on the examination as to whether a tonsillar swelling was found, I do not find any reliable explanation of the leucocytosis. This patient has an exanthema, which does not entirely exclude scarlatina; she might, therefore, also belong to Group III.

#### 36 CASES OF ASCERTAINED SCARLATINA (ONLY ONE COUNT).

Case No.	Diagnosis	Day of Disease	Temperature	Total count of leucocytes	Eosinophiles
65/18	Scarlatina	3rd	normal	19100	10½
90/18	"	3rd	37.9° C.	14800	7
633/18	"	3rd	39.2	18600	4
790/18	"	15th	normal	12000	9½
922/18	"	10th	normal	12400	6
989/18	"	4th	37.9	9500	8
27/19	"	6th	38	18600	7
159/19	"	8th	normal	12700	10½
258/19	"	3rd	normal	11500	7
265/19	"	5th	39	9900	5
459/19	"	5th	normal	10100	6
471/19	"	3rd	39	10300	3
552/19	"	7th	38	11900	7

Case No.	Diagnosis	Day of Disease	Temperature	Total count of leucocytes	Eosinophiles
555/19	"	3rd	38	14600	3
558/19	"	3rd	normal	13000	1
573/19	"	5th	38.4	11500	7
575/19	"	5th	normal	10600	8
581/19	"	3rd	37.9	14300	8
722/19	"	5th	normal	12100	6
759/19	"	6th	39	17500	8
782/19	"	9th	normal	10900	4
848/19	"	5th	38.2	16200	6
778/19	"	5th	normal	12200	5
901/19	"	9th	normal	15200	5
914/19	"	3rd	normal	14200	8
997/19	"	3rd	normal	10500	5
1166/19	"	4th	normal	10700	6
1184/19	"	11th	normal	10400	2
1274/19	"	3rd	normal	12000	4
1277/19	"	3rd	38.2	10900	2
44/20	"	2nd	normal	13200	8.8
61/20	"	4th	37.2	12500	5
116/20	"	15th	38	18200	4
1534/20	"	15th	normal	11300	4
1628/20	"	4th	normal	10100	2
117/21	"	3rd	normal	18800	6½

## SUMMARY OF CHAPTER VII.

1. In this material comprising 165 patients admitted to hospital for observation for scarlatina it appeared that 63 had a mild scarlatina while the others suffered from many different infections or intoxications, which gave rise to exanthemata or changes in the throat, causing them for a shorter or a longer period to be kept under observation for scarlatina.

2. Of the 63 scarlatina patients 49 had, during the first two to three weeks after admission, a somewhat varying leucocytosis, in every single patient at least once being above the range of error (i. e., above 12,000). Eleven patients had counts between 10,000 and 12,000, which may mean leucocytosis, but which also may result from a counting error. In three patients the count was between 9,000 and 10,000.

3. Only in about one-third of the above mentioned 49 cases was an increase of eosinophiles found during the first week of the disease.

4. In 58 cases which were not scarlatina there was no leucocytosis in the nonfebrile stage, while 28 cases of the same category showed a leucocytosis which could, however, in all cases be referred to a clinically traceable source.

In the total material comprising 165 patients only three cases had a leucocytosis, the causative factor of which could not be discovered.

5. In 13 cases out of 14, where the diagnosis of scarlatina was possible or probable, leucocytosis (and in certain cases eosinophilia) was found as a symptom which, in accordance with the results mentioned under the preceding items, seems to corroborate the diagnosis.

6. Of 36 cases of undoubted scarlatina in which only one leucocyte count was carried out during the first two weeks of the disease, a leucocytosis between 10,000 and 19,000 was found in 34 patients, and the contents of the blood of eosinophiles was between 5 and 10½ per cent in 22 cases.

#### CHAPTER VIII.

(a) The blood picture in patients with peritonsillar abscess and phlegmon and chronic tonsillitis.

(b) The blood picture in patients with simple tonsillar hyperplasia and chronic pharyngitis.

Leucocytosis in angina tonsillaris was mentioned in Chapter VII. It appeared from the tables that the leucocytosis often disappears at the stage of defervescence, while some cases show that this cannot be maintained as an absolute rule.

This lack of an absolute interdependence between temperature and leucocytosis already testifies that it is not the fever in itself, or rather the increased heart activity accompanying the increased temperature, which is the primary factor of the leucocytosis in angina tonsillaris. The differential count, with its special increase of neutrophile polynuclears, also indicates true infection leucocytosis, provoked by the inflammatory process in the tonsils. At the end of the same chapter a single case is mentioned in which leucocytosis was found to persist for a longer period in a nonfebrile stage, the case being not one of scarlatina. From the abstract of the casebook, it appears that there were pains in the throat and difficulties in swallowing, and the case was considered as one of angina tonsillaris with a protracted course; according to the symptoms it must be supposed that inflammatory processes, presumably small abscesses in the tonsils, persisted after the lacunar membranes had disappeared.

I was impelled to study whether chronic tonsillitis may be accompanied by an increase of leucocytes by reason of such isolated instances in the large material of patients under ob-

servation for scarlatina, in which a leucocytosis found in the nonfebrile stage makes the rule set up in Chapter VII, under item 4, less tenable, unless the inflammatory processes in the tonsils should afford a natural explanation of the leucocytosis, in connection with our knowledge of the clinical aspect of chronic tonsillitis. The available literature throws no light on this problem.

For the following reasons the nosologic term chronic tonsillitis has not to this very day been exactly defined; microscopic investigations, which have been carried on to a great extent in many institutes in the different countries up to this very day, fail to show any way of drawing any definite line between a healthy tonsil and a tonsil that is the seat of a chronic inflammation. Bacteriologic investigations, which also have been carried on to a large extent, give the same result: in nearly 80 per cent of apparently healthy tonsils which had not given any symptoms, even deep in the crypts bacteria were found of the same kind as those found in chronic tonsillitis. In the macroscopic examination we have, of course, a clinical help, as the chronic inflammation in the course of time, in most cases, at any rate, produces an alteration in the appearance of the tonsil; it assumes the characteristic irregular surface with irregularly distributed deep lacunæ alternating with projecting follicles of unequal form and size. To this comes, in most cases, that the tonsil becomes more fibrous in consistence, of which it is possible to convince oneself by a careful palpation. These macroscopic alterations, however, do not in reality tell us anything of the present function of the tonsil; they may indicate processes which have long ago taken place in tonsils which for many years have not been the seat of morbid processes. A somewhat more important objective symptom is an often periodically appearing edematous aspect of the tonsil, the latter being usually hypertrophic; this edema of the surface of the tonsil gives the characteristic impression of closed lacunæ, and through this purely mechanical condition, the swollen follicles causing relative blocking up of the lacunæ, a vicious circle no doubt comes into existence, as the retention at the bottom of the crypts tends further to maintain the inflammatory processes in the tonsils and the edema of the follicles.



Furthermore, the clinical test which consists in pressing detritus and pus out of the tonsils by exerting a moderate pressure on the organs between two spatulæ, affords no reliable information. There are individuals in whom neither detritus nor pus is found in many repeated examinations, or, at any rate, only in exceedingly small quantities, while numerous persons, in whose tonsils both detritus and pus are found over and over again, do not show any symptoms of a chronic tonsillitis. Therefore, I believe myself justified in saying—what also now is the general view—that the presence of detritus and pus—resembling liquid does not necessarily indicate a chronic tonsillitis, while it may indicate such.

It is reasonable to suppose that quite slight transitions exist between cases of chronic tonsillitis with exceedingly mild symptoms (e. g., a pricking sensation in the throat now and again, simultaneously with fatigue and indisposition, perhaps slight rheumatic pains somewhere in the musculature and now and then slight perspiration at night), and cases which cannot, even hypothetically be termed chronic tonsillitis.

In the otolaryngologic clinics, too, we meet with a large number of patients with small pale atrophic tonsils, out of which no pus or detritus whatever can be pressed by pressure with a spatula, and the consistence of which is not fibrous—patients in whom repeated attacks of rheumatic fever are no doubt of a tonsillogenic origin, since an angina tonsillaris usually was found to precede the pains in the joints.

In the last mentioned category of patients the diagnosis of chronic tonsillitis is hardly acceptable. The part played by the tonsils in such cases of rheumatic fever is, probably, that the crypts harbor the unknown virus which, on some provocation or other—e. g., cooling in consequence of a cold bath, or a sudden climatic alteration of temperature, attacks the organism without having produced alterations in the tonsils beyond those of the acute stage.

From the considerations stated above, it seems that we must seek other points of support for the diagnosis of chronic tonsillitis, and these, the most important points of support, are not found in the objective examination, but in the anamnesis. Information as to periodical acute angina, or only periodical pains in the throat and tenderness of the regional glands, fa-

tigue and indisposition to work, more or less quickly passing myalgias, perspiration at night and disposition to perspiration at the least effort, in certain cases a slight rise of temperature, and, still, some more or less marked symptoms which, in connection with those mentioned above, suggest that the diagnosis neurasthenia or phthisis pulmonalis incipiens almost outweighs the objective examination itself.

Peritonsillar abscess and peritonsillar phlegmon are phenomena which also belong to chronic tonsillitis far more frequently than to acute tonsillitis, and which afford visible evidence of the fact that, without special signs of infection, deeper lying suppurative processes may be found in a tonsil, processes which, on account of the edematous blocking up of the surface, make their way into the soft palate. And, finally, the finding of a true tonsillar abscess—for instance, a tonsil which presents a quite normally looking surface, although the objective inspection may prove that it is considerably enlarged—and out of which, at a certain spot, a quantity of pus, possibly amounting to the size of a kernel of a nut, may be pressed by means of spatulæ—should suggest to us that we may expect to find an excess of white corpuscles in certain cases and at certain stages of a chronic tonsillitis.

Case 1.—A patient, aged 16, with peritonsillar abscess, has normal temperature (36.8), but an accelerated pulse rate and a fair leucocytosis, the count increasing from 16,500 to 23,000 from one day to the other, while at the same time a slight rise in the temperature occurs (37.8). The differential count shows an excess of polynuclears. The monocytes are also present in a quantity somewhat above the normal. Incision evacuates considerable pus from a comparatively well defined cavity; the temperature now falls to 37.5 and the pulse to 86, in the course of half an hour, and the number of leucocytes decreases to about one-third of the previous value—i. e., to 8,100—without the differential formula being changed. During the following days only a little pus is evacuated by dilatation; this indicates that the abscess was well evacuated, and the number of leucocytes now approaches the normal, while also the percentage of polynuclears shows a decline.

Case 4 is a patient, aged 25, with a peritonsillar abscess, increased temperature (38.4) and pulse (84), and a consid-

erable leucocytosis of 17,700 with particular increase of the polynuclears. After the incision, by which the impression is gained that the pus is well evacuated, the figure for leucocytes falls in the course of three-quarters of an hour to about half its former amount without the formula being changed beyond the range of error. The pulse only falls to 82. Some infiltration persists during the following days, and in harmony with this the leucocyte count keeps somewhat above the normal—i. e., between 14,700 and 11,400.

The first case, especially, is of interest because the patient has a considerable leucocytosis in a nonfebrile stage; that the leucocytosis is provoked by the quantity of pus under pressure seems to be evidenced by the fact that it disappears when the pus is evacuated. It is true that the polynuclear formula is maintained during the great fall which takes place in the course of half an hour, and there is thus good reason to assume that the great decrease in the leucocytes is essentially due to changes in the heart activity, as the pulse at the same time falls to 86. It is, however, only in the course of the following twenty-four hours that the polynuclears reach their normal as an expression of the discontinuance of the infection.

In Cases 2 and 3, the patients have no well defined abscess, but peritonsillar phlegmon; with a slight elevation of the temperature, and with considerably increased pulse rate the patients have a very marked leucocytosis of 17,400 and 29,200, respectively, with a special increase of polynuclears. On incision only a small amount of pus is evacuated, while the tissue is seen to be highly infiltrated by pus. In accordance with these findings it also appears that we do not get any essential decline in the leucocytes, as also the formula remains unchanged. These two instances represent a type of cases of peritonsillar phlegmon in which the incision did not bring about the great decline in the number of leucocytes which took place in a number of cases with peritonsillar abscesses.

Cases 5, 6, 7, 8, 9 and 10 deserve to be specially mentioned; in these cases no peritonsillar abscess or phlegmon is found, but the patients suffer from a chronic tonsillitis, with more or less acute exacerbations; the symptoms were pains in the throat, especially on swallowing, tender angular glands, fatigue, indisposition to work, and, as far as some are concerned,

also perspiration at night. In several of these cases the temperature and pulse were normal, in other cases a slight rise of the temperature was found (37.6-37.9).

These patients have an undoubted leucocytosis during the acute stages. In some cases the excess of leucocytes is very considerable; thus in Case 6 a count of between 18,000 and 19,000 is found, although the temperature is normal. Cases 7, 8, 9 and 10 afford an opportunity of studying these relations both in the intervals between acute attacks, when there is no leucocytosis, and during the attacks when it appears that there is leucocytosis, as these patients were requested to meet for examination as soon as they felt the symptoms. In a considerable number of cases, on days when the patients in question complained of pains in the throat and other symptoms of an acute exacerbation in a chronic tonsillitis, I made sure by isolated tests that leucocytosis is a general and well marked "symptom" during these periods, the leucocytosis being even a finer indicator of the infection in the tonsils than both pulse and temperature.

When looking at Case 7 and Case 10, it seems as if the same effect may be produced as by the incision of the peritonsillar abscess, viz.: a fall in the leucocytes—only by evacuating the pus in the small tonsillar abscess by pressing with a spatula. That these small tonsillar abscesses are in reality not quite innocent phenomena seems to be evidenced by the well marked subjective symptoms of the patients. As for the objective symptoms, I shall be inclined to regard the leucocytosis found in these cases as a very important symptom of an infection injurious to the organism. In the practice of a laryngologist it happens numerous times that his patients comment on the wonderful improvement they feel in their condition after having had their tonsils pressed out, and I believe through these investigations to have got a clearer understanding of the diseases in question here.

In addition to these cases of chronic tonsillitis, characterized by intermittent acute periods as described above, a number of patients are seen at the otolaryngologic clinic, who have simple hyperplasia and who exhibit quite a different clinical picture, their only symptoms being, as a rule, the troubles

which are a direct consequence of the mechanic disproportion in their pharynx. It is not, however, denied that patients with simple hyperplasia show a marked disposition to develop the ordinary acute anginas with high temperature, and I am sure that there is a transitional form from this category to the above mentioned one. As far as the majority of these patients are concerned, it holds good that they do not present true symptoms of a chronic tonsillitis. In accordance with the above mentioned facts, I did not succeed in showing any leucocytosis in the latter category of cases, neither when the hyperplasia, as is often the case, was accompanied by a chronic pharyngitis or a chronic catarrh in the upper respiratory passages.

INVESTIGATIONS OF LEUCOCYTOSIS IN PATIENTS WITH PERITONSILLAR ABSCESS AND PERITONSILLAR PHLEGMON.

Case 1.—H. B., female, 16 years old. July 29. Complains of pain in right side of throat, especially on swallowing, and feels as if "the throat is thick." Suffering began three days ago. Temperature not measured at home. Objective: Right tonsil swollen, no visible pus. Soft palate very swollen just above tonsil. Redness of pharynx, larynx normal; stethoscopy of lungs, normal. Temperature 26.8, pulse 102, regular and vigorous. Leucocytes 16,500 (75.3, 0.2, 14.3, 10.2).

July 30. Swelling somewhat increased. Temperature 37.8, pulse 104. Leucocytes 23,000 (78.0, 0.0, 15.2, 6.8). At 10:15 incisio abscessus peritonsillaris. At 10:45 pulse 86, temperature 37.5. Leucocytes 8,100 (76.3, 0.1, 14, 8.7). At incision ample grayish yellow foul smelling pus was evacuated.

July 31. Temperature 37.5, pulse 84. Leucocytes 11,500 (65.0, 0.7, 27.1, 7.2). Dilatation with Lister's forceps caused a small amount of pus to be evacuated.

August 1. Still retention of some pus. Leucocytes 10,200 (64.7, 0.8, 25.0, 9.5).

Diagnosis: Abscessus peritonsillaris.

Case 2.—I. B., male, 30 years old. September 1. Comes to the free hospital clinic with pains in right side of throat. Duration, 3 to 4 days. Objective: Abscess and edema of left tonsil, the lacunæ of which are seen closed, without visible pus. Soft

palate projecting just above tonsil. Temperature 37.8, pulse 130. Leucocytes 17,400 (78.1, 0.1, 14.7, 7.1). At incision only little pus evacuated. Spots of pus seen in tissues of soft palate. Thirty minutes later, leucocytes 16,800 (77.0, 0.0, 16.3, 6.7).

September 4. Only slight projection. Leucocytes 12,300 (69.3, 2.0, 21.7, 7.0).

September 5, 9,500; September 6, 9,100.

Diagnosis: Phlegmon peritonsillaris.

Case 3.—Male, 30 years old, admitted October 12. Much harassed by large swelling of right side of pharynx. Large peritonsillar phlegmon in right side. Temperature 38.2, pulse 120. Leucocytes 29,200 (75.7, 0.7, 19.8, 3.8). Incision phlegmonis peritonsillaris. Thirty minutes later: 19,700 (78.8, 0.2, 18.1, 2.9). One and one-half hours after incision: 18,900 (75.5, 0.8, 18.9, 4.8). Pulse 80, temperature 38. Three hours after incision: 19,900 (78.8, 0.6, 18.1, 2.5). Ten hours after incision: 20,100 (77.9, 0.5, 19.4, 2.2). Pulse 102, temperature 38.3.

Diagnosis: Phlegmon peritonsillaris.

Case 4.—E. H. male, 27 years old, comes to the free hospital clinic. Pains in right side of throat. Temperature 38.4, pulse 98. Great projection of soft palate above right tonsil.

September 13. Leucocytes 17,700 (78.4, 0.2, 12.4, 9.0).

Incision of abscess with evacuation of a tablespoonful of grayish foul smelling pus. Three-quarters hour later: 8,500 (75.5, 0.3, 18.0, 5.5). Pulse 82.

September 24. Tonsil and adjacent part of soft palate gone down, but still rather considerable infiltration. Leucocytes 14,700.

September 25. Still some infiltration; 13,600. Temperature 37.5, pulse 74.

September 26, 14,300; September 28, 11,400.

Diagnosis: Abscessus peritonsillaris.

Case 5.—H. H., male, 24 years old. June 18. Complains of pains in throat since yesterday, when the temperature was 40 C. Tonsils swollen with lacunar plugs of pus. Angular glands tender. Temperature 38.3, pulse 110. Leucocytes 18,500 (80.1, 0.0, 15.3, 4.6).

June 19. 18,300 (77.1, 0.2, 12.7, 10.0). Temperature 39.3, pulse 104.

June 21. No plugs of pus in tonsils. Right tonsil only swollen. Left tonsil swollen and projecting. Temperature 38.3, pulse 98. Leucocytes 18,000.

June 22. Leucocytes 17,600; Temperature 38.1. By pressure with spatula pus evacuated behind the anterior palatine arch.

June 23. 16,500 (78.0, 0.3, 13.2, 8.5). Temperature 38.1. Moderate peritonsillar swelling around left tonsil.

June 25. 15,600. Temperature 37.8. Still some swelling around left tonsil.

June 27. 15,300. Temperature 37.9. By pressure with spatula on left tonsil abundant grayish pus evacuated.

June 28. 14,000. Temperature 38.1.

June 30. 7,100 (51.6, 4.0, 39.3, 5.1). Temperature 37.3. No abscess to tonsil. No pus.

Diagnosis: Angina tonsillaris, abscessus tonsillaris.

Case 6.—G. J., male, 23 years old. September 19. Pains in throat since yesterday. Redness and swelling of tonsils with follicular membranes.

September 23. Tonsils swollen, slightly edematous; no visible pus. Temperature 37.1, pulse 84. Leucocytes, 19,300 (74.3, 0.2, 15.5, 10.0). By pressure with spatula pus evacuated from both tonsils. Glands at both maxillary angles somewhat swollen and tender.

September 24. No membranes on tonsils, but they are tense and somewhat swollen. Leucocytes, 21,200 (78.4, 0.0, 13.4, 8.2.) Temperature 37.6, pulse 88.

September 25. Tonsils tense on palpation. By pressure with spatula pus evacuated from both tonsils. Leucocytes, 18,100 (74.0, 0.8, 14.0, 11.2).

September 27. Tonsils less swollen. By pressure with spatula small quantity of gas evacuated from both tonsils. Temperature 37.1, pulse 68. Leucocytes, 11,100 (70.3, 2.2, 17.4, 10.1).

September 28. 8,800.

Diagnosis: Angina tonsillaris with protracted course (abscessus tonsillaris).

Case 7.—Chr. P., female, 29 years old. February 27. Referred from medical polyclinic, suffering from pharyngitis acuta. States that for periods he has pains in the throat, feels tired and indisposed to work, suffers from perspiration at

night. Knows nothing of increases of temperature during these periods. Objective: Mucous membrane of pharynx somewhat hypertrophic with abscess of solitary follicles and side strings. Tonsils grooved with some detritus plugs, but no pus. Angular glands not tender. Cavum nasi, rhinopharynx, nothing abnormal. Stethoscopy of lungs, nothing abnormal. Temperature 37.0, pulse 66. Leucocytes, 6,100 (61.2, 3.0, 24.6, 11.2).

March 3. State unchanged. The objective findings as on March 27. Leucocytes, 6,700.

March 15. Has for a few days suffered from pains in the throat with mucoc secretion in throat. Has felt tired; perspiration last two nights. Temperature 37.1, pulse 68. Pharynx: Tonsils are normal, slight tenderness and swelling of angular glands on right side. Leucocytes, 13,100 (68.4, 2.8, 19.5, 9.3). Through pressure with spatula on right palatine arch liquid pus is evacuated from a small tonsillar abscess. One and one-half hours later, 6,500 (69.7, 2.4, 20.0, 7.9).

March 16. Nothing abnormal about tonsils; no pus pressed out. Feels well. Temperature normal. Leucocytes, 6,500.

March 17, 6,500; March 22, 6,100.

Diagnosis: Tonsillitis chronica (recurrent tonsillar abscess).

Case 8.—G. T., female, 26 years old. July 15. Complains of periodic pains in throat and mucous secretion. During these periods he feels indisposed, perspires easily, occasionally also in the night. Knows nothing of increase of temperature during these periods. Objective: Walls of pharynx somewhat red; tonsils of moderate size; scanty pus evacuated from left tonsil.

July 16. Complains of pains in left ear. Left tonsil slightly swollen. By aid of spatula, a little pus evacuated as yesterday. Tender angular glands on left side. Temperature 37.1, pulse 72. Leucocytes, 13,100 (72.3, 0.6, 17.1, 10.0).

July 18. No pus in tonsils. Temperature normal. Leucocytes, 9,100.

July 28. Has felt quite well. No sensation in throat. Tonsils natural. No pus pressed out. Leucocytes, 6,700.

September 10. Feels again a slight pricking in left tonsil and pains in left ear. Otoscopy, nothing abnormal. By pressure with spatula on left tonsil, pus evacuated, partly behind anterior palatine arch, partly from fossa supratonsillaris.



Slight tenderness at palpation of left angular gland. Temperature 37.3. Leucocytes, 10,300.

September 12. 10,500. Tonsils as on September 9.

September 14. 6,500. No pus.

September 16. 5,600. Some thin flowing pus pressed out from left tonsil. No subjective complaints; feels quite well.

September 18. 6,100. Still some pus in left tonsil, but no pains. No tenderness at palpation.

Diagnosis: Tonsillitis chronica with recurrent tonsillar abscess.

Case 9.—H. K., female, 27 years old. States to suffer from periodic pricking in throat and cough. During these periods patient feels indisposed, perspires easily. Is tired; knows nothing of increases of temperature. This condition has persisted for some years. Objective: Pharynx: Mucous membrane red, coated with mucus. Mucopurulent secretion in rhinopharynx. Tonsils of normal size. Nothing abnormal revealed by stethoscopy of lungs. Temperature normal. Larynx normal. Cavum nasi normal. Pulse 68. Tender angular glands on both sides. By pressure with spatula some pus is evacuated from left fossa supratonsillaris.

September 4. Leucocytes, 11,400.

September 5. 7,000. Some redness of throat. Still some pus pressed out from left tonsil.

September 6. 10,100. Pus pressed out from left tonsil.

September 8. 9,500.

September 10. 9,100.

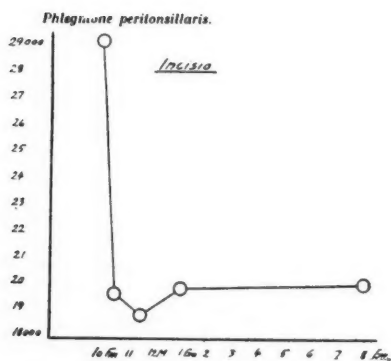
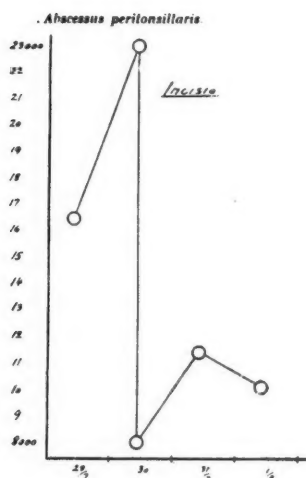
September 12. 10,000. Still pus pressed out from left tonsil.

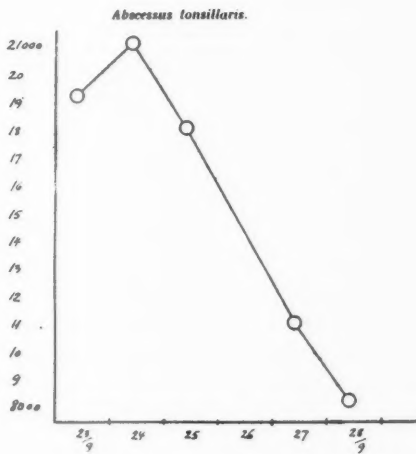
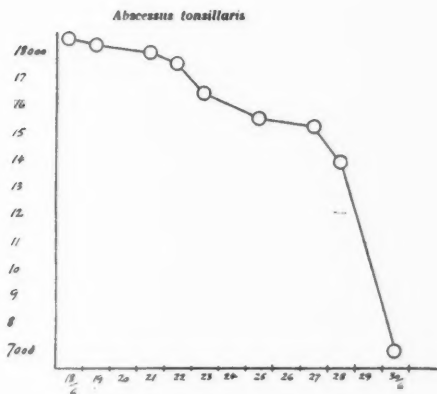
September 14. 10,500. Tonsils as on September 12. Temperature normal.

September 20. 7,500. Scanty thin flowing pus at left tonsil. Angular glands not tender. Feels well.

Diagnosis: Tonsillitis chronica (recurrent tonsillar abscess).

Case 10.—A. F. J., male, 25 years old. June 18. Complains of frequent pricking pains in throat; has often been confined to bed for some days on account of recurrent angina. Objective: Cavum nasi natural. Otoscopy, nothing abnormal. Stethoscopy of lungs, normal relations. Pharynx: tonsils hypertrophic with deep lacunæ. Temperature 37.4. Pulse 72. Leucocytes, 14,100 (74.3, 0.4, 17.3, 8.0). By pressure with spatula





pus pressed out from both tonsils. Tenderness at palpation on angular glands.

June 20. 6,500 (65.3, 2.0, 25.4, 7.3). No pus in tonsils, no tenderness.

June 24. 7,800. No pus in tonsils; no tenderness.

June 30. 9,000. No pus in tonsils, but from left fossa supratonsillaris a detritus plug the size of half a pea is pressed out. Some tenderness on tonsil, even on very careful palpation.

July 1. 5,400. Still detritus, but no pus.

July 24. States to have had pains in the throat since yesterday. The left tonsil increased. Tender, even at very careful palpation of tonsil. The corresponding angular gland tender. Temperature 37.5; no pus. Leucocytes, 12,000 (70.7, 1.9, 14.3, 13.1).

July 25. Still no pus to press out from increased left tonsil. Angular gland tender. Leucocytes, 13,100 (73.0, 2.0, 16.7, 8.3).

July 27. 13,100. By pressure with spatula pus evacuated from left tonsil; 30 minutes later, 10,100; 60 minutes later, 7,000.

August 13. Has had no trouble with throat. Tonsils not increased. By pressure with spatula thin flowing pus is pressed out from left tonsil and detritus from right tonsil. Leucocytes, 10,400.

August 19. Has felt indisposed for three days with some pains in throat. Measured temperature at home; no fever. Objective: By pressure with spatula on left tonsil some bad smelling grayish pus was evacuated from fossa supratonsillaris. Leucocytes, 14,100 (75.1, 0.8, 14.8, 9.3). Temperature 37.5, pulse 74.

August 21. Almost no pus in left tonsil. Leucocytes, 8,300. Still a little pus in left tonsil.

August 27. 7,900. Still a little pus in left tonsil. Feels well. Diagnosis: Tonsillitis chronica (recurrent tonsillar abscess).

#### CASES OF PHARYNGITIS CHRONICA AND HYPERTROPHIA TONSILLARUM WITHOUT INFLAMMATION.

Case 1.—Z. A., male, 17 years old. Admitted May 23 to the polyclinic. Tonsils hyperplastic; no pus to press out. No tenderness of angular glands. Temperature normal.

May 23, 6,100; 26, 7,200; 30, 5,900; June 2, 7,200; 4, 5,800.

Diagnosis: Hypertrophica tonsillarum.

Case 2.—C. E. J., male, 17 years old. Tonsils hypertrophic without pus; fauces red. She calls on the clinic on account of her large tonsils, which annoy her when swallowing. No pains in throat, no febrile periods, no perspiration a night.

July 3, 5,300; 5, 6,100; 9, 6,000; 11, 5,800.

Diagnosis: Hypertrophica tonsillarum.

Case 3.—A. J., male, 40 years old. Chronic pharyngitis with swelling of solitary follicles and redness of mucous membrane; suffers from a tickling cough; has been treated with caustic paintings for a longer time. Temperature normal. Nothing abnormal revealed by stethoscopy of lungs.

September 19, 5,500; 21, 5,600; 23, 4,900; 26, 5,900.

Diagnosis: Pharyngitis chronica.

Case 4.—O. J., female, 23 years old. Has never had angina. Has a "dry" sensation in throat and suffers from a tickling cough. Tonsils hypertrophic without pus. A few detritus plugs without decomposition. Fauces somewhat red. Temperature normal. Stethoscopy of lungs, nothing abnormal.

July 18, 6,000; 20, 7,500; 22, 7,100.

Diagnosis: Pharyngitis chronica. Hypertrophica tonsillarum.

Case 5.—M. J., male, 25 years old. Complains of cough from throat and pricking sensations. Has never had angina. No fatigue, no perspiration at night. Granular pharyngitis. Tonsils smaller than normal, if anything, with detritus plugs. No pus. No tenderness at palpation of tonsil; no tenderness of angular glands.

September 16, 8,700; 18, 6,500; 22, 7,500; 24, 8,100; 26, 8,000.

Diagnosis: Pharyngitis chronica granularis.

Case 6.—E. L. male, 20 years old. Pharyngitis chronica with swelling of side strings. Pricking in throat, a few detritus plugs; no tenderness of tonsils or angular glands.

September 9, 7,000; 11, 6,900; 13, 6,500; 15, 7,200.

Diagnosis: Pharyngitis chronica.

Case 7.—A. R., female, 45 years old. Has suffered from a cough for six weeks. Nothing abnormal found by stethoscopy of lungs. Slight redness of mucous membrane in throat and

larynx, where slight veiling of vocal cords is seen, and also swelling and redness of the false vocal cords.

July 16, 6,200; 18, 8,200; 20, 6,000; 22, 7,100.

Diagnosis: Pharyngitis chronica, laryngitis subacuta.

Case 8.—A. S. male 39 years old. Suffers from much cough. Temperature normal. St. pulm.: normal relations. Slight redness of mucous membrane on posterior wall of pharynx and slight diffuse redness of mucous membrane in larynx. Tonsils of normal size; no pus. Temperature normal.

July 17, 7,600; 19, 7,700; 22, 6,800; 28, 8,100.

Diagnosis: Pharyngitis chronica, laryngitis chronica.

#### SUMMARY OF CHAPTER VIII.

1. Patients with peritonsillar phlegmon and abscess have leucocytosis; in a single case of those examined the leucocytes reached nearly 30,000, while in the remaining cases counts between 15,000 and 23,000 were found.

2. Incision brings about a rapid and great fall in the number of leucocytes when pus is evacuated (abscess), while no fall is seen by incision in the case of phlegmon.

3. Tonsillar abscess frequently gives rise to a leucocytosis of from 13,000 to 20,000 (often without any increase of the temperature). The existence of this leucocytosis in connection with the subjective complaints afford a more reliable indication of the infectious state of the patient than the relations of the temperature and the pulse.

4. In patients with chronic tonsillitis pus is often found in the tonsils—apart from the acute exacerbation—without any increase of the number of leucocytes, while leucocytosis is present in the acute stage, even without increase of the temperature.

5. Patients with simple tonsillar hyperplasia and chronic pharyngitis have no leucocytosis.

#### BIBLIOGRAPHY.

- Appelberg, R.: Om Canceranämiens Natur. Disp. Helsingfors, 1919.  
 Arneht: Die weissen Blutkörperchen bei Infektionskrankheiten. Jena, 1904.  
 Aschenheim: Naturforscherversammlung. Münster, 1912.  
 Baastrup, Skat: Hæmatologiske Undersøgelser af Scarlatinapatienter. Ugeskrift for Læger, Juli, 1918.  
 Bauer: Deutsche medic. Wochenschrift, No. 44, 1921.

- Becker: Om Blodbilledet hos Botriocephalusbårare. Akademiska Afh. Helsingfors, 1915.
- Bennecke: Monatsschrift Fischer, Jena, 1909.
- Bloch, E.: Deutsche med. Wochenschrift, No. 29, 1903.
- Bonsdorff, A. von: Finska Läkarsällskapets Handlingar. Vol. 54, 1912.
- Bonsdorff, A. von: Den normale Leukocytformeln, Finska Läkarsällskapets Handl. Vol. 56, 1914.
- Boycott: British Medical Journal, 1903.
- Breuer: Berliner klin. Wochenschrift, No. 41, 1902.
- Bruhn-Fähräus: Nord. Med. Archiv. No. 15 og 17, 1897.
- Bruns, Liefmann and Mächel: Münch. med. Wochenschrift, 1905, p. 253.
- Buckler: Münch. Med. Wochenschrift, No. 2, 1894.
- Bucklers: Cit. efter Opie: American Journal, 1904, p. 477.
- Carstanjen: Cit. efter Gundobin. Jahrbuch für Kinderheilkunde. Bd. 52, 1900.
- Christiansen, Max: Disp. Paris, 1923, p. 134.
- Ellerman and Erlandsen: En ny Leukocyttællingsteknik Hosp. No. 48, 1909.
- Ellerman and Erlandsen: Psykiske Forhold som Aarsag til Svingninger i Leukocyttallet. Hosp. No. 13, 1909.
- Ellerman and Erlandsen: Om Leukocyttælling og Inhomogenitet. Ugeskrift for Læger, No. 32, 1910.
- Ellerman & Erlandsen: Bidrag til Studiet af de fysiologiske Svingninger i Leukocyttallet. Hosp. 1910, No. 37, og 37.
- Elzholz: Wiener klin. Wochenschrift, No. 32, 1894.
- Ehrlich: Farbenanalytische Untersuchungen. Berlin, 1891.
- Escherich and Schick: Scharlach. Wien, 1912.
- Felsenthal: Archiv für Kinderheilkunde. XXV.
- French: Cit. efter Naegeli: Blutkrankheiten. Berlin, 1919.
- Freudenthal: Hospitalstidende, 1924.
- Fuchs-Rosenthal: Wiener med. Presse. Bd. 45, 1904.
- Galambos: Fol. hæmatologica. No. 13, 1912.
- Glaser: ref. efter: Klinische Wochenschrift, 1922, No. 29.
- Goodall and Paton: Journal of Physiology. Bd. 33, 1905.
- Grawitz: Deutsche med. Wochenschrift. 1910, No. 29.
- Gram, H. C.: Disputats. København, 1921.
- Grek and Reichenstein: Wiener klin. Wochenschrift. 1908, No. 14.
- Gundobin: Die Besonderheiten des Kindesalters. Berlin, 1912.
- Halla: ref. efter Grawitz: klin. Pathologie des Blutes. 1911.
- Hasselbalch and Heyrdahl: Kgl. danske Videnskabernes Selskabs. Forhandlingar No. 5, 1907.
- Hayem: Leçons sur les modifications du sang. Paris, 1882.
- Hirt: cit. efter Naegeli: Die Blutkrankheiten.
- Hoffman: Zeitschrift für klin. Medicin. 1897. Vol. 33.
- Holzer and Schilling: Zeitschrift für klin. Medicin. 1922. p. 302.
- Hutinel: La clinique. April, 1908.
- Japha: Jahrbuch für kinderheilkunde. Vol. 52, 1900.
- Japha: ref. af A. von Bonsdorff: Finska Läkarsällskapets Handl. Bd. 54, No. 7.
- Jørgensen, Carl: Hospitalstidende, 1913. No. 40, og. 41.
- Jørgensen, Gustav: Disp. København, 1915.
- Jørgensen, Gustav: Hospitalstidende, 1917, p. 1117, og. 1142.

- Jungmann: Deutsche med. Wochenschrift, 1921, No. 44.  
 Karnitzki: cit. efter Gundobin.  
 Kisch: Deutsche med. Wochenschrift. 1922, No. 46.  
 Kjer-Petersen: Disp. Aarhus, 1905.  
 Knöpfelmacher: Wiener klin. Wochenschrift, 1893.  
 Kotschetkow: ref. efter: Escherich and Schick: Scharlach, Wien, 1912.  
 Kroll: Diss. Basel, 1913.  
 Körner: Lehrbuch der Ohren-, Nasen- und Kehlkopfkrankheiten. Wiesbaden, 1918.  
 Labbé-Besançon: Traité d'hématologie. Paris, 1904.  
 Laucner: Jahrbuch für Kinderheilkunde, 1916.  
 Lerenski: Fol. hæmatologica. Vol. 9, p. 12.  
 Limasset: ref. efter Naegeli: Die Blutkrankheiten.  
 Limbeck: Zeitschrift für Heilkunde. Vol. 10, 1890.  
 Lindström and Tallquist: Citeret efter Appelberg: Disp., 1919.  
 Malassez: De la numération des globules rouges du sang. Paris, 1873.  
 Malassez: Citeret efter Reinert.  
 Memmi: ref. efter Naegeli.  
 Meulengracht and Gram: Hæmatologisk Teknik. København, 1922.  
 Moleschott: Wiener med. Wochenschrift, No. 8.  
 Moleschott: Cit. efter Reinert.  
 Moro: Archiv für Kinderheilkunde. 1910, Vol. 40.  
 Müller, Sven: U. f. L., 1917.  
 Naegeli: Die Blutkrankheiten und Blutdiagnostik. 1919.  
 Nicolas and Courmont: Archiv de med. exp. et d'anat. path. 9, 737, 189.  
 Nicolas and Courmont: Comptes rendus soc. de biol. 1898.  
 van Noorden: ref. fra Pappenheim.  
 Ostendorf: Fol. hæmat. 1923.  
 Pappenheim: Grundriss der hæmatol. Morfologie. Leipzig, 1921.  
 Perlin: ref. efter Gundobin.  
 Philipp: Presse médicale. 1921, No. 48.  
 Pohl: Archiv für exp. Pathologie und Pharmacologie. Vol. 25, 1889.  
 de Pury: Virchow's Archiv. Bd. VIII.  
 Putzig: Zeitschrift für Kinderheilkunde. IX, p. 165, 1913.  
 Rabinowitz, D.: Diss. Basel, 1912.  
 Ragosa: Folia hæmatologica. XIX, 1915.  
 Ramsey: Fol. hæmat. 1915, p. 269.  
 Reckzeh: Zeitschrift für klin. Med., XLV.  
 Reinecke: Diss. Halle, 1889.  
 Reinert: Die Zählung der Blutkörperchen. Leipzig, 1891.  
 Rieder: Beiträge zur Kenntniss der Leukocytose. Leipzig, 1892.  
 Richet: De l'anafylaxie alimentaire. Soc. de biol., Jan., 1911.  
 Rosenstern: Jahrbuch für Kinderheilkunde, Vol. 69.  
 Rud: Ugeskrift for Læger. Maj, 1924.  
 Sabrassez: cit. efter Naegeli.  
 Sandelin: Akademiske Afhdl. Helsingfors, 1916.  
 Salzberger: Diss. Freiburg, 1909.  
 Schiff and Stransky: Deutsche medicin. Wochenschrift, No. 44, 1921.  
 Schulz: Deutsches Archiv für klin. Medicin. Vol. 51, 1893, ref.  
 Seligman and Dudgeon: Lancet, 1902, Vol. 6, p. 21.  
 Sirensky: Fol. hæmatologica, VI, p. 175.



- Sonnenburg: Perityphlitis. Leipzig, 1913.  
 Sørensen, S. T.: Disp. København, 1876.  
 Terhola: Archiv für Gynæcologie. Vol. 103.  
 Thoma: Virchow's Archiv. Vol. 87, p. 201-209.  
 Thomsen, Oluf: Hospitalstidende, 1906, No. 9, og 12.  
 Tilleston and Locke: Journal of Infect. Diseases. 1905. Vol. II.  
 Tornow: Diss. Berlin, 1895.  
 Türk: Klin. Untersuchungen bei Infektionskrankheiten. Wien, 1898.  
 Türk: Vorlesungen über klin. Hæmatologie. Wien u. Leipzig, 1912.  
 Wagner: Cit. efter Sørensen.  
 Welcher: Cit. efter Sørensen.  
 Wernstedt: Nordisk med. Archiv II Ad. 1910, p. 43.  
 Widal, Abrami and Brissaud: Comptes rendus de la soc. biol., 1913,  
 p. 429 og 502.  
 Widal: Presse médicale, 1920, No. 90.  
 Virchow: Cellularpathologie. 1871.  
 Winternitz: Centralblatt für innere Medicin. 1893.  
 Zappert: Zentralblatt für klin. Medicin. No. 19, 1892.  
 Zappert: Zeitschrift für klin. Medicin. No. 23, 1893.  
 Zarniko: Krankheiten der Nase. 1910.

XIX.

OTOGENIC PARALYSIS OF THE ABDUCENS, WITH  
SPECIAL MENTION OF ISOLATED PALSY  
ASSOCIATED WITH IRRITATION OF  
THE GASSERIAN GANGLION\*

INCLUDING

I. A REPORT OF TWENTY-SIX UNPUBLISHED CASES; TWO FROM THE WRITER'S PRACTICE, AND TWENTY-FOUR FROM THE RECORDS OF OTHER OTOLOGISTS; ALSO A THIRD CASE OF THE AUTHOR'S, REPORTED FIFTEEN YEARS AGO.

II. A TABULATION OF FINDINGS, NOTED IN A SERIES OF ONE HUNDRED AND SEVENTY-TWO PATIENTS, INCLUDING THE TWENTY-SEVEN ABOVE, AND ONE HUNDRED AND FORTY-FIVE OTHERS RECORDED IN LITERATURE MAINLY SINCE 1910.

III. A BIBLIOGRAPHY OF ALL REFERENCES RECORDED TO DATE.

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Paralysis of the sixth cranial nerve arising in the course of suppurative otitis, or in the presence of an intracranial extension of otogenous disease, has been recorded in literature for almost half a century. It was not, however, until less than two decades ago that a considerable number of these cases was collected, analyzed and thoroughly discussed.<sup>1</sup>

Three years later another group of similar cases was assembled and an analysis<sup>2</sup> of those which had appeared in the literature to that date was made. Since that time many reports with observations in various phases of the subject have been published, but no effort has been made to parallel the work noted above, or to bring it up to date.

The writer has endeavored to collect all cases appearing in the literature, which had not been included in the first two

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\*Candidate's thesis, American Laryngological, Rhinological and Otological Society, recommended for publication by the council.

publications mentioned. The number occurring in the practice of any one man is limited. Fourteen years ago a well known metropolitan otologist saw two in one year, but has had none since. Five is the largest number distinctly reported in American publications, observed and treated by a single aurist; while in foreign journals, the maximum for a number of years has been six.

It is probably less rare than it seems, for doubtless many cases have been seen and correctly diagnosed but not reported; many others met with, but not diagnosed; while not a few are found in literature under other titles.

When this paralysis does occur in the course of otogenic suppuration, the symptoms are usually alarming, and the otolaryngologist who beholds his first case in that stage should have some acquaintance with the subject, or have immediate access to textbooks discussing this problem. This has not been possible in the past, for, despite the fact that otogenic palsy of the abducens was brought forward prominently twenty years ago, and has been discussed at intervals ever since, it is of recent date that reference to the subject can be found in any work on otology, exceeding casual mention. The paralysis may be isolated, combined, bilateral, homolateral or contralateral.

#### ISOLATED ABDUCENS PARALYSIS.

Of isolated paresis or paralysis of the extraocular muscles, that of the external rectus is most frequent, forming more than one-third of all such cases. This is due to the fact that the sixth nerve supplies no other muscle as well as to the unusual anatomic position and relations of this nerve. The etiologic factors upon which this is dependent are quite numerous, owing to the long and exposed course of the abducens nerve at the base of the brain, its relation to adjacent structures, and the great number of pathologic processes to which these are susceptible.

#### CHARACTER OF PARALYSIS AT DIFFERENT POINTS OF THE NERVE TRACT.

Although a lesion may affect any part of the nerve tract from its cortical center to its peripheral ending in the muscle, the resultant paralysis varies materially with the point attacked.

Affection of the intracerebral portion always induces a conjugate and never an isolated palsy. Involvement of the right nucleus produces paralysis of the right externus associated with paralysis of the left internus, again an interference with conjugate movement. Therefore, in dealing with isolated palsy of the externus, we need to consider only affections of the root fibers emerging from the nucleus and the nerve itself in its course along the base through the sphenoid fissure and in the orbit to its final destination. This constitutes, according to Llandoldt, the peripheral part of the nerve, which he divides naturally into fascicular, basal and orbital.

Fascicular or root palsies may arise from lesions (tumor or hemorrhage) in the pons, which not infrequently simultaneously induce a contralateral hemiplegia. In its course at the base of the brain the abducens is more likely to be affected by external influences than essential disease of its own structure. The principal one is pressure. This may be due to hemorrhage, exudation in meningitis, new growth of the pons, cerebellar fossa or petrous bone. Tumor of the cerebrum, but more especially of the cerebellum growing toward the base, may produce it as a distant symptom. Traumatism is a potent cause; fracture of the base on account of the intimate relation of the sixth nerve to the apex of the petrous being particularly likely to produce it; indeed isolated palsy of the externus may be at times the only symptom of such fracture.

Isolated orbital palsies do occur, but are less common. At the entrance to the orbit all of the ocular nerves are so closely related that multiple palsies are more likely to occur. This is also true of the orbital cavity. They may arise, however, from local cellular inflammation, tumors of small size, traumatism such as blows and inflammatory reaction in the muscle or nerve from exposure.

#### OCURRENCE IN VARIOUS DISEASES TO BE NOTED IN DIFFERENTIAL DIAGNOSIS.

Isolated paralysis of the abducens has been observed in many affections of the brain and spinal cord; tabes, cerebro-spinal syphilis, multiple sclerosis, acute bulbar paralysis, acute ascending paralysis, syringomyelia, hemispheres, tumors, abscess, hysteria; disorders of excretory and secretory organs;

chronic nephritis, diabetes mellitus and insipidus; intoxications, chronic alcoholism, lead, drug and ptomain poisoning; acute infectious diseases; diphtheria, influenza, erysipelas, cerebrospinal meningitis, herpes zoster ophthalmicus, typhoid fever, malaria, rheumatism, acute anterior poliomyelitis, epidemic encephalitis, paralytic dementia, myasthenia gravis, vascular change, including arteriosclerosis, aneurysm, occlusion and rupture of vessels, chlorosis, traumatism, lumbar anesthesia.

Köllner's<sup>3</sup> study of 250 cases of paralysis of the externus, 107 of which were isolated and 143 in combination with other ocular palsies, is the largest and most complete the writer found while making a search of the literature in 1909. If one of a similar character has appeared since then he has overlooked it. This report gave lues, hereditary and acquired, and tabes as the cause in 28 cases of the 107 isolated palsies, or a little less than 25 per cent, and the cause in 84 cases of the 143 combined palsies, or more than 50 per cent. Cushing,<sup>4</sup> discussing Köllner's study, says:

"Doubtless in the large majority of the syphilitic cases the nerve was directly involved in a gummatous meningitis. However, judging from the fact that in none of Köllner's series was the diagnosis confirmed by a subsequent section, it is not improbable that a number of the cases, classified as lues, were more properly tumors. This at least would conform with our experience with the clinical diagnosis of intracranial lues."

In Köllner's series, otogenous disease was the cause of two of the isolated, and of thirteen of the combined paralyzes, thus less than 2 per cent of the isolated cases arose in the course of otitic affection, though, of the combined palsies, 6 per cent were of otogenic origin.

#### ESSENTIAL STRUCTURES IN RELATION WITH THE PETROUS PYRAMID.

A study of the otogenic palsy of the abducens of other of the nerves supplying the extraocular muscles necessitates mention of the structure adjacent or contiguous to the petrous pyramid, which may be directly or indirectly affected by this infection.

As is well known, the dura acts in a dual capacity; first, the outer layer serves as a periosteum, nourishing the internal

walls of the cranium, the inner as a supporting membrane for the cerebral tissues. In the main, these two layers are inseparable, but in certain locations they separate to form the walls of the cranial sinuses. Of these sinuses, the lateral, the superior, and inferior petrosal, and the cavernous, come in contact with some part of the petrous bone, while the jugular bulb is in contact with the floor of the hypotympanum.

The cavernous sinus, by reason of its position at the apex of the pyramid, and its peculiarly intimate anatomic association with the third, fourth, fifth and sixth cranial nerves, any or all of which may be affected in the course of a suppuration of the temporal bone, merits a short description.

This sinus is a channel for venous blood, situated between the supporting and periosteal layers of the dura on the side of the body of the sphenoid bone, and extending from the central end of the sphenoid fissure in front to the apex of the petrous bone behind. It is the most complicated of all the sinuses formed by the dura mater and in length is about 2 cm., while its width is about 1 cm., being almost quadrilateral on cross section. Anteriorly it is a continuation of the ophthalmic vein and posteriorly it ends in the superior and inferior petrosal sinuses. It is in direct communication with the cavernous sinus of the opposite side, also with the basilar plexus.

The sinus contains in the outer wall from above downward, and from before backward, in the order named, the oculomotor, the trochlear and the ophthalmic division of the fifth nerves. These are separated from the blood of the sinus by its lining membrane. The internal carotid artery and the sixth nerve traverse the sinus, the nerve being below and lateralward.

External to the cavernous sinus a continuation of the separation of the dural layers encloses a space over the apex of the petrous pyramid, called by the older anatomists Meckel's Cave. This contains the Gasserian ganglion.

Spalteholz's description of this region is quite clear. (*Hand Atlas of Human Anatomy*, 1923, Vol. III, p. 709.)

"The dura mater completely lines the depth of the fossa hypophyseæ, spreads out above the same between the tuberculum sellæ and the upper margin of the dorsum sellæ as the diaphragma sellæ, and there presents only a small opening,

foramen diaphragmatis (sellæ), for the passage of the infundibulum. It then runs lateralward on each side and stretches out from the processus clinoideus anterior and from the processus clinoideus posterior to the angulus superior pyramid (lateral from the impressio trigemini); thence it passes, behind, downward to the clivus and to the facies posterior pyramidis, lateralward, however, obliquely downward to the middle cranial fossa (lateral from the foramen rotundum and foramen ovale), and thus helps to enclose a large space in which are situated the a. carotis interna, the sinus cavernosus, the n. oculomotorius, n. trochlearis, n. trigeminus, along with its ganglion semilunare and its branches, as well as the n. abducens."

Again, on the posterior surface of the pyramid, this dural separation encloses, over the aqueductus cochleæ, the saccus endolymphaticus. Of the arterial blood supply we are concerned with the internal carotid which passes through its canal in the petrous bone, thence through the medium lacerated foramen into the cranial cavity, where it first curves upward, then forward, lying in the carotid sulcus of the sphenoid bone on the inner wall of the cavernous sinus, covered by the lining membrane of the sinus. In the carotid canal it is surrounded by a plexus of veins, which receive tributaries from the tympanum; this plexus opens into the cavernous sinus and internal jugular vein, forming a direct connection between the two. The carotid also gives off a branch to the tympanic mucous membrane. In its passage through the canal it is also associated with the carotid plexus from the cervical sympathetic as well as its lymphatic plexus.

In addition to the internal carotid must be mentioned those branches of the basilar artery which may be in relation to the abducens nerve in its course between the clivus and the pons.

#### COURSE OF THE SIXTH NERVE.

"The superficial origin of the sixth nerve is in the groove at the junction of the lower border of the pons and medulla, a little lateral to the pyramid, where the emergence of both sixth nerves is but a few mm. apart. After leaving the surface of the brain stem the course of the nerve runs upward and lateralward, following the lower or ventral surface of the pons

for about 15 mm. It then pierces the dura mater over the sphenoid bone, a little above the junction of the basilar process of the occipital bone with the sphenoid, and turns forward in the interval between the apex of the petrous part of the temporal bone and the posterior clinoid processes of the sphenoid, passing in this situation under cover of a ligament, which connects the bony processes mentioned." (Morris, Human Anatomy, 1896.)

It enters the floor of the cavernous sinus lying below and to the outer side of the internal carotid artery, and eventually reaching the outer wall of the anterior part of the sinus, enters the orbit through the sphenoid fissure lying above the ophthalmic vein and below the third, fourth and ophthalmic branch of the fifth nerve. Leaving the fissure it passes between the two heads of the external muscle, which, after entering its ocular surface, it supplies.

THE ANATOMIC DATA GIVEN ABOVE, EXCEPT WHERE QUOTED, HAS BEEN TAKEN FROM VARIOUS STANDARD ANATOMIES.

The intimate anatomy of the course of the nerve after piercing the dura to its entrance into the cavernous sinus, has been made plain by Dorello, whose description is appended. Wheeler<sup>5</sup> gives a very good description of his dissection of the same region, illustrated by most excellent plates. Vail<sup>6</sup> made a series of dissections of eight cadavers and from these drew sketches which graphically pictured Dorello's work, as Dupuy<sup>7</sup> has done also by his own dissections. The writer's dissection of two specimens followed the same lines with similar findings.

Dorello's description follows (a rather literal translation of pp. 170-171 from Gradenigo's article<sup>1</sup>):

"The sulcus petrosus superior disappears four or five mm. from the point of the temporal bone and a bony process of varying form and strength takes its place, the point of which is directed upward, inward and forward. i. e., toward the processus clinoides posterioris. This process, which Dorello named the spina sphenoidalis, is represented as the continuation of the posterior lip or margin of the sulcus petrosus superior; the sinus petrosus superior bends somewhat outward from the spina toward the front to discharge into the sinus cavernosus. Inwardly from the spina there is a depression in a rather deep plane, corresponding to the point of the tem-



poral bone, then follows the sutura petrosphenoidalis and finally the outer margin of the lamina quadrangularis of the sphenoid bone, which presents a variable form and strength. In many cases this describes the form of a third part of a circle, whose upper extremity is formed by the point of the clinoid process posterior. Sometimes it is differentiated into two parts by a bony process, which somewhat below the clinoid process posterior bends outward and is called by Dorello the accessory process of the posterior clinoid. In this way there is formed a sinus between the spina sphenoidalis and the clinoid process posterior, the cavity of which extends upward and outward, which, however, by the presence of soft parts is metamorphosed into a bony fibrous canal, and especially through the interposition of a very strong fibrous bundle, which is called the ligamentum petrosphenoidale (Gruber).

"Moreover, to understand the anatomic relation of this ligament it is necessary to bear in mind the art and manner of the attachment of the tentorium cerebelli in its anterior part. This structure, as you know, is crescent shaped with an outer border by which it is attached, and is bounded by a free inner aperture socalled of Pacchionii. The bundles of fibers of the tentorium which lie nearer to the inner border are superficial to the others, run in a sagittal direction, somewhat outwardly from the clinoid process posterior, as far as the clinoid process anterior, attach themselves to these and strengthen the outer and upper margin of the sinus cavernosus. The outer bundles of the tentorium, on the contrary, which pass from outward within, are inserted, first on both margins of the sulcus transversus of the occipital bone, then on those of the sinus petrosus superior. The more medial bundles are arranged nearly transversely, pass under the sagittal tissue of the inner margin of the covering, intersect these crosswise, join the clinoid process posterior and aid in the formation of that fibrous layer which protects the ganglion Gasserii.

"The bundles lying deepest can be dissected as special formations and they form the petrous sphenoid ligament. This passes from the spina sphenoidalis of the upper margin of the temporal bone, enters the outer margin and the posterior surface of the lamina quadrangularis of the sphenoid bone somewhat below the clinoid process posterior at the site of

the accessory clinoid process posterior, which appears as nothing less than the bony origin of the petrous sphenoid ligament. Between the inner part of the upper margin of the petrous bone—the part under the outer margin of the lamina quadrangularis of the sphenoid bone—and the petrous sphenoid ligament is a small nearly three cornered space with the point outward and the base inward. In this space lies the abducens, also the sinus petrosus inferior at the orifice into the sinus cavernosus. The abducens lies on the lower wall of this space, usually near to the outer angle, and sometimes it is confined to the angle which the sphenoid spine forms with the upper edge of the pyramid, and it is here somewhat flattened from above downwards.

"The sinus petrosus inferior runs very obliquely from the rear to the front and from outward inward until it reaches the abducens, which runs in like manner from behind forward, but from within outward, about centimeter below the point of the pyramid directly in front of where it enters the dura mater. After the meeting of both structures, the direction of the sinus becomes straight, approaching more nearly to the sagittal plane lying above the nerve and enters with it into the osteofibrous canal mentioned above. Here, however, the relations between both differ according to the side which the abducens occupies. If this occupies the outer angle of the canal the sinus lies on the inner side of it; but by the nerve lying more toward the median line, the sinus lies above it and covers it entirely except the under side. Immediately after leaving that space the nerve makes a slight bend, enters into the sinus cavernosus and passes in close connection to the carotid artery."

#### ROUTES OF INFECTION.

Having described briefly some of the anatomic considerations which have to do with the Gasserian ganglion and the sixth nerve, it may be well to consider the various ways and different paths by which a suppurative otitis media can extend its infection to these structures. It is obvious that these routes will be identical with those which are followed in an intracranial extension.

These are (1) direct; (2) indirect.

(1) Direct Routes: Since the Gasserian ganglion comes in contact with the petrous pyramid where it lies in the tri-

geminal impression near the apex of the bone, and the abducens comes in actual touch when it rests in the outer angle of Dorello's canal, against the sphenoid spine of the petrous, direct transmission must occur by extension of the tympanic process inwardly through the pyramid to its apex.

Various routes through the petrous to reach the apex have been described; as a matter of fact, in extensively pneumatized bone, this transfer can pass through any contiguous group of cells and in this way any number of routes could be followed. That the type of temporal bone must have much to do with the ease or difficulty of purulent transfer goes without saying. Where very extensive pneumatization has taken place cells may be found distributed everywhere, and an especially large cell may at times be found at the point of the pyramid. One of Wheeler's<sup>5</sup> plates illustrates a temporal bone of this type. Collet<sup>8</sup> shows a specimen in which this apical cell was 12 mm. long by 10 mm. in width, and 10 mm. in height. In such case the peritubal cells may be large and transfer will pass readily by them to the cells at the apex.

In this type of bone the protective lamina has been encroached upon and is naturally thin, specimens of the writer showing the bone of the trigeminal impression to be no thicker than paper. When such a favorable anatomic arrangement occurs; that is, large cells underlying the trigeminal impression and adjacent to the region of the sphenoid spine, affection of the bone passes readily to the dural floor of Meckel's Cave (a pachymeningitis) and thus affects the Gasserian ganglion. In like manner can the abducens be influenced through an osteitis, an osteomyelitis, or, for that matter, by any inflammatory process at the apex sufficiently violent to produce a reactive inflammation in the tissues beyond. Since the result of a transferred inflammation may vary from a simple hyperemia to a violent necrosis, is it not possible, in some cases at least, that this process may be limited to an edema sufficient to compress the nerve and impair function, which goes on to rapid subsidence after free drainage has been established at the original site of infection?

The labyrinth offers another direct route, although much less commonly in an acute than in a chronic suppuration, by way of the oval or round windows, or necrosis of the prom-

ontory or prominence of the external semicircular canal; from the labyrinth to the cells about it and thence to the apex. There is also an indirect route via the internal auditory meatus or the ductus and saccus endolymphaticus, or caries of the petrous. Passage along the carotid canal must also be mentioned.

(2) Indirect Routes: Here again the type of structure of the temporal bone doubtless has direct bearing. If the bone is dense and nonpneumatic the immediate transfer of a purulent infection from the tympanum is impeded. Wittmaack's work is of special interest in this type of bone, which has failed of normal pneumatization; is it not possible that the large and abnormal vascular connection from the tympanum is impeded? Wittmaack's work is of special sinuses and the meninges (to be seen in some of his specimens) may be the deciding factor in a primary thrombosis of the first three structures.

Venous channels from the tympanum to the petrosal sinuses, the carotid plexus and the jugular bulb, congenital bony dehiscences in the floor of the hypotympanum, or roof of the epitympanum, as well as minute canaliculi for the passage of vessels and nerves, all serve as channels for transmission of the purulent process or its effects indirectly to the structures in question. This may be through the petrosal sinuses and jugular bulb by thrombophlebitis, or in passing to intracranial structures above the tegmen tympani, by pachymeningitis or extradural abscess. When the antrum and mastoid cells become part of the infective process, transmission intracranially may follow by any one of the usual routes. Finally, the lymphatic system must be mentioned as a route for the passage of infection.

#### LITERATURE.

The first mention of abducens paralysis, complicating suppurative otitis and associated with Gasserian pain,\* was by Gradenigo, who, in 1904, discussed this question, reporting at the same time five cases of his own and a number of others he had collected.

In a further publication, three years later, the same author tabulated the records of all cases appearing in the literature to that date, fifty-seven in number; to these was added a dis-

\*As a syndrome.

cussion of eight cases in considerable detail and a general consideration of the various points involved.

From this study he drew the following conclusions:

There exists a typical clinical picture which is essentially characterized by an acute purulent inflammation of the middle ear and by intensive pains, especially in the temporoparietal bone region of the diseased side, and also by a paralysis of the abducens of the same side. In exceptional cases the clinical picture can be produced through acute exacerbation of a chronic purulent inflammation of the middle ear. In about half of the cases accessory symptoms can be present, which depends on damage of the trigeminus and the oculomotorius or of the cerebral membranes. Complications arise sometimes, as mastoiditis and circumscribed and restricted extradural lesions on the sigmoid sinus.

Generally a complete recovery takes place, and but seldom does death result with symptoms of a diffuse purulent leptomeningitis. The pathologicoanatomic process consists in a diffusion of the pus infection of the drum cavity to the pyramid apex through the peritubal air cells and the carotid canal. The abducens is affected at the apex of the pyramid close to its passage into the dura mater. We have to deal with a circumscribed osteitis at the pyramid apex and eventually with a corresponding pachymeningitis. The extradural location of the disease explains its generally favorable course. In individual cases, however, there occurs a circumscribed serous or diffuse purulent leptomeningitis.

"Not all cases of paralysis of the abducens, which occur in the progress of otitis, belong to the described clinical picture. Unquestioned forms of these depend on various pathologic causes. Among those occurring most frequently are the extradural deep abscesses on the roof of the pyramid, which extend to the apex of the same. Also there occurs a diffuse osteomyelitis of the covering of the air chamber of the temporal bone, which reaches the petrous point. Cases of this kind naturally do not belong to the type and accordingly the prognosis and therapeutics of the same appear different."

If, in discussion of this subject, frequent *referenec* is made to Gradenigo's work, it is because he made the first and largest distinct contribution to its study.

The Gradenigo syndrome, as noted above, has for its basis suppurative otitis media, severe frontotemporoparietal head pain, or other pain in the area of distribution of the fifth nerve, and paresis or paralysis of the abducens of the same side; the Gasserian irritation and paralysis of the sixth nerve resulting from extension of the middle ear infection through the peritubal air cells and the carotid canal to a large cell at the apex of the petrous pyramid (of a large celled pneumatic temporal bone), resulting in an osteitis, circumscribed, pachy- or leptomeningitis, edema of structures about the sixth nerve in Dorello's canal, with consequent pressure on the sixth, inducing paresis or paralysis.

In the accompanying analysis the presence or absence of pain is noted in 131 cases—only twice was pain recorded as not present. Of 129 times present, 121 times it was severe, and but eight times moderate or slight. The pain was characterized variously as agonizing, unbearable, intense and severe; localized as frontal, temporal, parietal, facial, side of head, auricular, occipital, general head, neck, orbital, peri-orbital, in the eye and eyelid, upper and lower jaw, teeth, and in the ear, seldom at one point, usually two or more of the points mentioned being affected.

Since this series covers all forms of otogenic involvement, it seems evident that severe homolateral head pain in the regions supplied by the trifacial nerve is commonly associated with abducens palsy of otitic origin; so that the so-called Gradenigo triad cannot be limited to the one anatomicopathologic process outlined by him. This seems to be true, whether the paresis of the sixth is so slight as to merit a simple note of its presence, in the course of alarming intracranial disease, or the paralysis complete, and an outstanding symptom of an otherwise mild illness.

With these facts in mind, one sees that while all cases published under the name of the Gradenigo syndrome will present this triad of symptoms, many of them may be due to an entirely different pathologic extension of the tympanic sup-puration, and therefore do not belong to this classification.

Of the fifty-eight authors who gave a pathologic diagnosis, nineteen attributed the affection of the sixth nerve to a circumscribed pachymeningitis at the apex of the petrous; nine

to a serous or protective meningitis; four to an inflammatory edema at the apex of the petrous; seven to edema of the brain with increased intracranial pressure; sixteen to toxic neuritis; four to extension of inflammation from lateral sinus and bulb via inferior petrosal sinus to region of the sixth nerve; one to isolated thrombosis of the superior petrosal sinus; two to extradural abscess; one to encephalitis; four to abscess at apex of petrous; one to hemorrhage into brain substance. Forty-three of these represented pressure in some form. This seems most likely to occur in many of the cases through pressure on the sixth nerve in Dorello's canal, which has been described above. This is especially true of the cases of circumscribed pachymeningitis or an inflammatory edema at the apex of the petrous.

In serous meningitis of high degree, or in cerebral edema, it seems probable that pressure may be exerted at other points in the course of the abducens, with a resultant paresis or paralysis.

Cushing,<sup>4</sup> in a study of paralysis of the abducens occurring in association with brain tumor, gives an illuminating picture of the relation of the basilar artery to the abducentes as they course along the pons with a clearly demonstrated hypothesis as to the causation of certain palsies, by the constricting influence of these vessels on the sixth pair.

Quoting from his article:

"The branches of the basilar artery (the only vessels which we need consider in relation to the sixth pair) pass transversely across the pons in a direction more or less at right angles with that taken by the abducentes. The two branches which come into direct relation with the nerve are: (1) The A. cerebelli inferior anterior, which passes outward and backward across the crus cerebelli, to be distributed to the anterior border of the under surface of the cerebellum, anastomosing with the posterior inferior cerebellar branch of the vertebral; and (2) the A. auditiva interna, which may either arise as a separate branch from the main basilar trunk or else through the bifurcation of the A. cerebelli inferior anterior."

He discussed the superficial relation of the arteries to the nerves and showed that in some instances the artery may be superficial on one side and the nerve superficial on the other.

Summarizing, he says:

"It seems fair to say, therefore, (1) that in the usual topographic relationship the arteries are superficial and cross the nerves in such a way as to render constriction possible; (2) that in the majority of the fatal cases in which diplopia (particularly in association with a conjugate strabismus) has been recorded, the pons was grooved and the abducentes constricted by the arteries; (3) that these symptoms were much more common in the cerebellar cases, though not alone restricted to them; (4) that the cerebellar lesion, though often so remote as to render unlikely any possible direct affection of the nerves themselves or of their centers, nevertheless, owing presumably to swelling and enlargement of the hindbrain, produced such a stretching of the arteries around the brainstem as to constrict and groove the nervous tissues, much as though a rubber band had been placed around them; (5) that in cases of extreme constriction the nerves may be found degenerated and replaced by scar tissue at the point of constriction; and, finally, (6) that the 'distant' symptom of a convergent squint may disappear after the successful removal of a cerebellar tumor whose situation was too remote to have directly affected the peripheral neurones of the sixth pair.

"If on this basis, abducens palsies may in some cases be attributable to arterial constriction, they may in a sense be regarded as one of the general manifestations of pressure or tension; a convergent squint, furthermore, like each of the other so-called pressure symptoms, may have a certain localizing value as speaking in favor of a lesion of the hindbrain."

"The so-called syndrome Gradenigo has been observed as a complication of otitis media without abscess or other demonstrable cerebral lesion. I have seen several instances of this combination, in each of which the lesion was attributed to a cerebral edema, for all of the patients showed headache, vomiting, and a low grade of choked disc. These edemas are presumably akin to the meningitis serosa of Quinke, and in most of our cases a lumbar puncture, or a subtemporal decompression has not only largely relieved the pressure symptoms, but has led to a rapid subsidence of the diplopia. The fluid in all of these cases has been sterile. It is not improbable, therefore, that disturbances of this kind may be ascribed to some swelling



and edema of the brainstem, which had brought about the same constricting effect on the part of the cerebellar arteries that we have described as existing in cases of brain tumor."

Ruttin<sup>9</sup> gives the following explanation for the occurrence of papillitis in sinus thrombosis, in which he also refers to the presence of an abducens palsy under certain conditions:

"Judging by our observations we must assume that there is a true engorgement and that an engorgement of the cavernous sinus is the cause of papillitis; this engorgement is brought about by regurgitation of the blood from the superior and inferior petrosal sinuses.

"If we now assume that the regurgitation from the superior and the inferior petrosal sinuses toward the cavernous sinus is the cause of the papillitis, then among all otogenous intracranial complications, sinus thrombosis in respect to the operative exclusion of the sinus jugularis region should give the highest per cent of papillitis. This supposition also seems to be supported if systematic investigations are instituted. Of our thirty cases, eight had papillitis. Six of these cases developed papillitis only after jugular ligation and the removal of the sinus; in one case there had, unfortunately, been no examination of the eyes, and in another case the papillitis had existed before the ligation. The period of its development varied from two days to a month after the operation. In four of our cases, serous meningitis may be absolutely excluded.

"As to why papillitis should appear in one case of sinus thrombosis and not in another, we can at present form only an anatomic conjecture, for we know that: (1) the sinus connections are variable; (2) often where there is a thrombosed sinus, good drainage undoubtedly still exists out of the cavernous sinus, either through the superior petrosal sinus via sinus into the mastoid veins, or through the inferior petrosal sinus via bulbus into vena condyloidea; (3) the different development of the sinuses and the jugularis on the two sides is also not without its influence on these conditions.

"In any case these anatomic conditions probably play a more important part in the development of papillitis, in sinus thrombosis with its extreme one sided effects, than, for instance, in cerebellar abscesses or chronic hydrocephalus with equal pressure upon all four petrosal sinuses."

Auerbach and Alexander,<sup>11</sup> in explaining increased intracranial pressure in sinus thrombosis discuss the anatomic variations in the size and relations of the dural sinuses, with the findings of various anatomists.

Eagleton<sup>11</sup> (Brain Abscess R. 129) reports a case in which ligation of the left internal jugular vein in lateral sinus thrombosis resulted in complete venous stasis, with an associated choked disc, retinal hemorrhages and blindness. Autopsy showed the free right lateral sinus to be about one-half as large as the left thrombotic sinus. The ligated jugular vein was the main vessel for the return of blood from the intracranial cavity. He further explains an associated acute internal hydrocephalus by increase of the cerebrospinal fluid from irritation and from interference with discharge of venous blood through the veins of Galen, its discharge through the arachnoid villi being at the same time prevented by thrombosis of the longitudinal sinus. The increased cerebrospinal fluid with diminished outflow resulted in an acute distension of the ventricles in addition to the venous stasis.

Twenty-seven times sinus thrombosis has been an added complication. Twenty-one times the palsy of the sixth seemed to be definitely associated with the thrombosis. Of these, ten times it followed ligation of the internal jugular vein; once it arose coincidently with a sinus thrombosis, following injury to and packing of the sigmoid sinus during mastoidectomy; three times it appeared in association with clinical signs of sinus thrombosis from four to seven days after a mastoidectomy (two of these had ligation and resection of the internal jugular vein and removal of the thrombus, the third had sinus uncovered, found thrombotic, but was not operated on; all three recovered); twice it was present with other symptoms of intracranial disease at first examination, in both cases the internal jugular vein was ligated and resected, and the thrombus removed; twice it appeared four weeks after mastoidectomy, of these one was associated with epidural and perisinus abscess and sinus thrombosis, the other with isolated thrombosis of the superior petrosal sinus; once it occurred in the presence of thrombosis of both cavernous, both lateral, and one inferior petrosal sinus in company with cerebrospinal fluid under high pressure; the last case arose after five days of acute illness

with symptoms of sinus thrombosis; early operation and removal of the clot followed.

One other case had an associated thrombophlebitis, with high degree of papilledema and other symptoms of increased intracranial pressure, which possibly belongs with the twenty-one mentioned above.

Of fifteen of the number with sinus thrombosis, thirteen reported papilledema, usually of considerable degree, while in two there was no eyeground change.

In Ruttin's<sup>9</sup> series of thirty cases of sinus thrombosis, six developed papilledema following ligation of the internal jugular vein, although but three developed at the same time an abducens paresis.

It is noteworthy that in this series ten out of twenty-one of the sixth nerve palsies in sinus thrombosis should have followed ligation of the internal jugular vein.

On the basis of the studies quoted it seems reasonable to conclude that ligation of this vessel results in venous stasis with cerebral edema and increased intracranial pressure, and that sinus thrombosis can produce similar results; so that compression of the abducens in Dorello's canal, or in its course up the pons by the branches of the basilar artery, seems the clearest explanation for that group of cases associated with sinus thrombosis and especially for those following ligation of the vein.

Of seventeen extradural abscesses, eight seem to be definitely causal of the paralysis. All of these latter followed softening or necrosis of bony tissue, so that a dural inflammation may be credited as extending from the smaller abscesses and affecting the sixth by swelling and pressure. In the large ones direct extension to the region of the nerve was noted during operation or at autopsy in four cases. Two accompanied sinus thrombosis and one was present with a sixth nerve palsy on the opposite side.

There were six temporal lobe abscesses. One ruptured spontaneously with immediate death and no autopsy; one encapsulated; the capsule adherent to the tegmen was accompanied by a bilateral sixth palsy, due to increased intracranial pressure; one was encapsulated with a stalk and drained spontaneously through an attic fistula, dural extension of inflammation being

the probable cause of paralysis; in one the palsy arose when the abscess was enlarging and intracranial pressure was much increased; the fifth had no relation to the palsy. There were three cases with cerebellar abscess; death resulted in two, the third, which recovered, was associated with sinus thrombosis and extradural abscess.

Sixteen cases are recorded with the finding during operation or at autopsy of a pneumatic petrous bone, with cells extending to the apex of the pyramid and containing pus. A very large cell in this situation, which drained for a month after operation, is reported by Sullivan.<sup>12</sup> Wilkinson's<sup>13</sup> case at autopsy disclosed an abscess cavity at the apex of the pyramid measuring 16 mm. in length by 12 mm. in height. Eklund's<sup>14</sup> case had very large pus filled cells about the middle ear opening of the eustachian tube; curetment of these was followed by suppurative meningitis and death. Caries or necrosis of the petrous pyramid was noted five times.

This group, with diseased cells well toward the apex of the pyramid, or with caries or necrosis of its anterior, superior or posterior wall, meets all requirements for direct extension of inflammatory process to both the Gasserian ganglion and sixth nerve.

Although the list enumerated accounts for quite a number of the paralyses, the majority remain without explanation other than deduction from the associated conditions and symptoms.

An anatomic condition favoring the extension of infection from the middle ear is a temporal bone of cellular or pneumatic type. Of the total number of cases, seventeen were not operated on; the character of structure was not stated in seventy-three; of the others, twenty-five were diploic or sclerotic, and sixty-four were of the large or small celled pneumatic type, many of these with very extensive distribution of cells. Thus, in eighty-nine cases almost 72 per cent were anatomically favorable for the diffusion of purulent infection either toward the apex or to the antrum and mastoid. Twenty-eight per cent were largely of the sclerotic or eburnated type, the sequence of chronic mastoiditis. This condition carries with it favorable factors for an intracranial extension of disease.

An intracranial condition favoring compression paralysis is increased pressure of the cerebrospinal fluid. In fifty-three

cases, where the pressure or absence of increased cerebrospinal fluid pressure was recorded, thirty-four times it was increased, while nineteen times the pressure was normal. In a number of cases the pressure was very high, and this would account for another group of the palsies. This, of course, indicates a protective meningitis, the exciting cause of which likewise might bear on the development of this paralysis.

Nine times no ear operation whatsoever was performed. Eight times simple incision was done. Of these one was incision of an otitis of the external auditory canal; the other seven were incisions of the tympanic membrane.

Of the nine without surgical procedure, two died from fulminating meningitis; another died from rupture of a brain abscess a few hours preceding the time set for operation; the third died while being prepared for operation; spontaneous perforation of the tympanic membrane occurred in the other two.

The eight cases in which spontaneous perforation alone, or incision of the tympanic membrane, resulted in final recovery from the abducens palsy, form part of the group to which Gradenigo<sup>1</sup> refers as typical of his syndrome. Of the two in which there was spontaneous perforation only, in one perforation occurred the second day of otitis, and the paralysis three days later, disappearing in three months; in the second, perforation took place the eleventh day, and the palsy eight days later, disappearing likewise in three months. Four cases with intact membrane were incised respectively on the second, fourth, and two on the eighth day of otitis; the paralysis appeared respectively on the twentieth, seventeenth, fourth and seventh days of otitis, disappearing respectively after thirty days, thirty-one days, and the last two in five days. The final two perforated spontaneously, but also had repeated incisions, the palsy in one arising on the twentieth day of otitis; in the other almost three months after ear symptoms arose, in both cases passing away weeks later. In this group is to be noted the appearance of the paralysis from four days to three months after the onset of otitis, and its disappearance in from five days to three months after its appearance.

There were thirty-one deaths, or 18 per cent. The terminal cause in twenty-four was suppurative meningitis, a number

of times fulminating in type; in two cerebellar abscess; in two temporal lobe abscess; in two encephalitis; in one erysipelas. Autopsies were done in ten cases and not done in twenty-one.

In the ten autopsies but four gave findings relative to disease of the temporal bone. One showed caries of the posterior wall of the pyramid. In the second an abscess cavity deep in the petrous, from which pus extended, had ruptured through the petrobasilar suture. The third showed an organized abscess in the apex of the pyramid. Wilkinson's<sup>13</sup> report of the autopsy in his case is given in detail; that relating to the petrous bone follows:

"On stripping the dura from the base of the skull, a thin layer of bone was detached from the apex of the petrous pyramid, disclosing a smooth walled abscess cavity. No direct communication was made out between this cavity and the collection of pus within the meninges. The dura was loosened from the bone in the neighborhood of the cavity, and particularly at the back of the cella, and some pus was tracking beneath the dura in this situation, though no direct communication could be discovered between this tract and the large collection of pus in the right middle fossa. The brain was kept for further examination, and the left temporal bone with the adjoining part of the basisphenoid and basioccipital was removed.

"The cavity in the apex of the pyramid measured 16 mm. in its long diameter and 12 mm. from above downwards. The carotid artery was exposed for about one-half inch in the anterior wall. Externally and posteriorly the cavity was bounded by the compact bone enclosing the internal auditory meatus. Below, the firm ligaments binding the petrous to the basioccipital formed the floor. A section was made parallel to the axis of the pyramid traversing the mastoid and tympanic cavity. It showed clearly the track of the infection from the tympanum by way of the cells extending from the anterior part of the tympanic cavity above and below the eustachian tube to the carotid canal, and thence to the area of spongy or cellular bone lying at the apex of the pyramid behind the carotid canal and internal to the internal auditory meatus. The lateral sinus was quite normal, as was the labyrinth.

"Further examination of the brain revealed nothing other than diffuse basal meningitis."

The value of the autopsy findings as to pathology of the pyramid in this series is rather less than that of the findings at operation, noted above. However, enough material has been developed to show that in a certain group of cases diffusion of the purulent infection from the tympanic cavity is direct to the cells at the apex of the pyramid, and thence to the ganglion and nerve.

According to this analysis, otogenic abducens paralysis appears most frequently in the first two decades of life, 51 per cent; the third and fourth decennials come second with 33 per cent; from forty years to sixty years, 15 per cent; and from sixty years to seventy-two years, less than 1 per cent.

As to sex, seventy-six males and fifty-five females. As to the side affected, right sixty-two, left fifty-six; bilateral six, and contralateral side, fourteen.

The onset is usually sudden and frequently the palsy is complete. Sometimes a paresis appears, which develops gradually into complete paralysis, as in the cases of Dench,<sup>15</sup> twelve days; Clark (Case R 10), six days; the writer (Case R 3), six days.

As to time of appearance, in eighty-nine cases where it was stated exactly, seventeen were in the first ten days; thirty-seven came in from the tenth to the thirtieth day, and twenty-nine in from one to two months; six after two months; so that, generally speaking, about 61 per cent arose during the first month. This is in marked contrast to Gradenigo's<sup>1</sup> series, in which out of thirty-seven cases, twenty, or about 56 per cent, developed between twenty and fifty days after onset of otitis; ten of them between forty-two and fifty days.

As to time of disappearance, in seventy-six cases, where the time was stated exactly: in the first ten days, nine; from ten to thirty days, fifteen; from thirty to sixty days, thirty; two months or more, twenty-two; 31 per cent clearing within thirty days, 40 per cent between one and two months, and 29 per cent after two months. These figures are more in accord with Gradenigo's,<sup>1</sup> the large majority clearing within three months.

One hundred and nine cases made complete recovery; ten cases were much improved, most of them practically well, while three remained unimproved at the time of report. The prognosis for recovery from the paralysis is extremely good, 95

per cent of this group of 122 having made either complete or almost complete recovery; it is indeed much better than prognosis for life, since out of this group of 172 cases, thirty-one, or 18 per cent, proved fatal. In Gradenigo's<sup>1</sup> series 7 per cent died, and in Perkins'<sup>2</sup> analysis,  $11\frac{1}{2}$  per cent resulted in death.

As a rule, this form of paralysis is homolateral. However, out of 124 cases it was contralateral fourteen times, and bilateral six times. Roy,<sup>10</sup> in 1921, reported a contralateral case of his own and gave a brief resumé and discussion of all others he had found in the literature, a total of ten. He includes in this list five cases purely contralateral, including his own, and five cases with bilateral abducens paralysis. From this group three contralateral and two bilateral appear in the writer's series, leaving two contralateral and three bilateral, which will be included and discussed in this analysis.

Taking up briefly the cases of bilateral paralysis, six of this series and three from Roy's.<sup>10</sup>

In two cases there was very extensive sinus thrombosis, choked disc, optic neuritis and increased cerebrospinal pressure, sufficient to account for bilateral palsy. One case was associated with extradural abscess and protective meningitis with cerebrospinal fluid under very high pressure; the paralysis improved rapidly after draining the abscess, but especially after repeated lumbar puncture—again an evidence of increased pressure as the cause. In another case temporal lobe abscess, with increased intracranial pressure, could be causal. The fifth case had bilateral scarlatinal otitis, left mastoidectomy, double sixth palsy. This case, of course, could have had cause in each temporal bone for the local palsy. The sixth case had forced adduction of both eyes with increased vertigo. The brief history does not read like a simple bilateral abducens palsy, but has, to some extent at least, the appearance of convergence spasm. The last three cases give nothing in the report to aid in making a diagnosis, unless an abscess in the neck muscles could have some influence. Of this a note will be made later.

As noted above, there were fourteen contralateral palsies in this series, with two to be added from Roy,<sup>10</sup> a total of sixteen. It is of especial interest to observe that eight of these were



associated with sinus thrombosis, six of them having followed ligation of the internal jugular vein.

Roy's<sup>16</sup> case exhibits that slow development with destruction of bone, characteristic at times of the streptococcus mucosus capsulatus. It apparently came on about the time of operation, and one can explain in such a case a homolateral palsy, but the associated symptoms give no clue to its contralateral character. Of the seven cases remaining, three were associated with symptoms of meningitis, with cerebrospinal fluid under high pressure (one of these had extradural abscess), sufficient to account for a homolateral lesion.

Quadri's<sup>17</sup> case might easily be of the homolateral group, and has but one associated point, which might indirectly have to do with its contralateral character, and that is the abscess in the neck following mastoidectomy. The other three case abstracts gave nothing in the way of a detailed report.

Otogenic abducens palsy contralateral in type is a very rare occurrence, this group of sixteen cases doubtless representing the large majority of all such palsies published. That out of more than 270 published reports of otitic paralysis of the sixth nerve, a relatively rare occurrence, about 6 per cent should be contralateral, indicates its very unusual character.

The fact that eight of these cases were associated with sinus thrombosis leads to the thought that the cervical phlegmon in Quadri's<sup>17</sup> case, and the abscess in the neck in Wheeler's<sup>5</sup> case of bilateral palsy may be of importance in the pathology.

It seems possible in both cases that an abscess in the region of the large vessel carrying away intracranial blood, could by pressure as well as toxic transfer lessen its outflow, and thus produce an additional intracranial factor of mild degree but of the same character operative in those cases with frank sinus thrombosis; especially might this be true if the vein on the opposite side was very small, as recorded in Eagleton's<sup>11</sup> case.

In attempting to explain these contralateral palsies, no study is complete until the accessory nasal sinuses, especially the sphenoids, have been demonstrated to be without disease.

The relation of the oculo-orbital nerves, including the abducens to the sphenoid sinus, was investigated and public report of the findings was made at about the same time by Sluder<sup>18</sup>

and Onodi.<sup>19</sup> Onodi<sup>20</sup> states (reference to all nerves except the sixth is omitted):

"The thin translucent bony wall of the sphenoid sinus was in direct contact with the abducens for a distance of 7, 10, 11, 12, 13 and 20 mm. In one case the wall of the sinus of the right side was in direct contact with the trunk of the sixth nerve on the left side. This preparation shows a great asymmetry of the sphenoid sinus. The right sphenoid sinus is 30 mm. broad and 28 mm. long, while the left sphenoid sinus appears as a small cavity 10 mm. in height and 12 mm. in breadth, in the neighborhood of the left optic nerve. The right sphenoid sinus extends to the left side and comes into contact with the trunk of the left abducens, and for a small distance the right abducens."

In addition he found in twelve specimens a close relationship (the bony wall being translucent and thin) between the sphenoid sinus and the abducens, eight times; homolateral in seven; contralateral in one.

"This fact, observed by us, gives the anatomic explanation of the contralateral paralysis of the abducens nerve in disease of a sphenoid sinus of the opposite side."

Frenel's<sup>21</sup> cases show graphically the importance of this relationship and the difficulty of differential diagnosis, when both the ear and the sinuses are involved. I. A male, nineteen years of age, left otitis ten days, intracranial symptoms (chills and vomiting), contralateral paralysis of the abducens. Mastoidectomy gave no explanation. At autopsy empyema of left sphenoid sinus was discovered. II. Case of chronic otitis, meningitis developed. Radical operation with exposure of both fossa revealed no pathology; bilateral paralysis of externi followed, later protrusio bulbi and chemosis (cavernous sinus thrombosis). Necropsy brought to light empyema of the sphenoid sinus. In each case simultaneous involvement of both sphenoid sinus and tympanic cavity, or possibly pansinusitis with involvement of the tympanic cavity, facilitated the infection of the sphenoid sinus in the interior of the cranium.

Sixty-one times in this series a predisposing cause of otitis is recorded. Setting aside twenty acute exacerbations of a chronic otitis, there were forty-one following influenza, head colds, sore throat, measles, scarlet fever, pneumonia, puerperal

sepsis and acute general disease. Influenza and head colds made up thirty of this group, a striking indication of a need for study of the nasal sinuses.

De Lamothe<sup>25</sup> speaks of three theories in the causation of a contralateral palsy: reflex, anatomic and serous meningitic. He regards the reflex theory as untenable, and after examination of a number of skulls dismisses the anatomic. He holds to a serous meningitis, with equally distributed pressure, when the more fragile nerve will suffer first. He suggests that the opposite sixth may be more delicate, and thus be affected, in this way developing a contralateral paralysis.

In explaining the paralysis on the opposite side, there are several possibilities, which, to the mind of the writer, seem feasible.

1. Latent disease (so-called) of the ear on the side of paralysis. An acute bilateral otitis at the onset, the one side remaining active, but the other side, after spontaneous perforation or incision of tympanic membrane, apparently healing, but actually developing a low grade extension to the tip with sixth palsy of that side, the process going on to spontaneous healing without further ear symptoms on that side, just as it occurred in some of the cases of unilateral otitis reported without operation. This condition could have occurred in Ersner's case (Case Report 4), but in view of accompanying symptoms is most unlikely.

2. Coincident homolateral disease of an asymmetric sphenoid sinus which is in direct contact with the sixth nerve on the opposite side. Onodi's<sup>20</sup> theory, illustrated by Frenel's<sup>21</sup> Case I above.

3. Coincident contralateral disease of a sphenoid sinus in contact with the affected nerve.

4. Pressure by branches of the basilar artery on the abducens in the case of meningitis serosa, or cerebral edema, causing increased intracranial pressure.

This condition, as we have seen, has been productive of homolateral palsies. In the contralateral group it seems possible to take into account Cushing's<sup>4</sup> theory of pressure by the branches of the basilar artery on the abducentes in the presence of meningitis serosa, or cerebral edema. He pointed out that occasionally the artery was superficial on one side,

while the nerve was superficial on the other, or an anomaly of arterial distribution left one nerve free, or the artery pierced the nerve, leaving part free from compression. In either case, if the nerve on the opposite side was compressed, and the nerve on the affected side was free, or partly free, a contralateral paresis would result.

5. The possibility in certain conditions of the nerve becoming more resistant on the affected side.

Comment has been made above on the occurrence six times of a contralateral paralysis following ligation of the internal jugular vein in the presence of sinus thrombosis, with the production of cerebral edema. It seems unlikely that so large a group could be dependent on the anatomic variations described by Cushing,<sup>4</sup> which are of themselves not frequent. It is well known that the intracranial structures, including the nerves, can adapt themselves to increasing conditions of pressure for a considerable time, provided this pressure is of slow development. Striking examples of this are given by Cushing<sup>4</sup> in the case of brain tumor.

With this in mind, the question follows whether in the case of a sinus phlebitis developing slowly to thrombosis, a gradually increasing edema and pressure, more marked on the diseased side and due to retardation of venous circulation, may not arise, and during this evolution the homolateral sixth nerve gradually accommodate itself to changed conditions. Then, when ligation of the vein follows, and the entire venous outflow depends largely on the opposite venous channel, may not the full force of edema and pressure now evolved find the sixth nerve opposite the diseased ear in a less resistant state, resulting in a consequent contralateral paresis.

6. Asymmetry in the size of Dorello's<sup>1</sup> canal, as determined by its bony margin.

Dorello<sup>1</sup> pointed out the presence of a projection from the lateral margin of the lamina quadrangularis of the sphenoid bone, a short distance below the posterior clinoid process, which he named the accessory posterior clinoid process. When this is present the petrosphenoid ligament, which covers in Dorello's canal, is attached to the sphenoid spine of the petrous externally and to the accessory posterior clinoid process internally. Frequently it is absent and then the ligament covering in the

canal has its attachment to the posterior clinoid process. This anatomic variation makes a very considerable difference in the size of this canal in different specimens.

Plate II, a photograph of a specimen of the writer's, shows very clearly (arrow IV on the left side) the accessory posterior clinoid process, to which the petrosphenoid ligament attaches itself. On the right side, arrow I points to the groove in which the sixth nerve lies, and just beyond the sphenoid spine; the point of arrow II is lying on the accessory posterior clinoid process. Dorello's canal lies beneath and between the two. The plate shows quite clearly (the skull itself more so) how very small the canal is on both sides. At the same time the canal on the right side can be seen to be considerably smaller than that on the left.

A second specimen of the writer's shows the type of Dorello's canal exhibited by Plate III, in which the accessory clinoid process is absent, and the ligament is attached to the posterior clinoid process, thus making, as can be seen, an extremely large canal, extending, in fact, almost a half circle. In the skull the canal on the left side is at least one-third smaller than that on the right.

These variations demonstrate the possibility of pressure developing a paralysis of the sixth nerve in the smaller canal. If that is contralateral, then a sixth palsy of the opposite side results.

The same anatomic variations may have somewhat to do with homolateral palsies. For example, in several cases, where exactly the same clinical symptoms are apparent, a sixth palsy occurs in one case and not in another, it seems quite possible that, other things being equal, a large canal may be present in one and a small one in the other.

Pathogenic organisms were recorded forty-six times, including the streptococcus, pneumococcus and staphylococcus alone; also mixed streptococcus, pneumococcus and staphylococcus, Klebs-Loeffler bacillus, influenza bacillus, colon bacillus, and two cases where the culture produced no growth. The streptococcus appeared not typed thirteen times, the mucosus capsulatus eleven times, the hemolyticus four times, and in mixed group seven times, or thirty-five times in forty-six. The pneu-

mococcus was reported five times; in pure culture five times, but not typed, and in mixed groups seven times, or twelve times in forty-six.

In this series the streptococcus was present in 76 per cent of the cases reporting an organism. There still seems to be some discussion by pathologists as to the exact placing of the mucosus, whether in the streptococcic or pneumococcic group.

MacCallum says that

"Pneumococcus, type III, sometimes called streptococcus mucosus, gives rise to the most severe, treacherous and destructive form of otitis media."

Loughran,<sup>22</sup> speaking of the streptococcus mucosus capsulatus, says:

"There is no doubt of its virulence and insidious tendency for the production of late complications; that in meeting this infection experience has taught that we are dealing with an organism whose power of rapid and extensive destruction of the mastoid bone may easily be able to combat the best efforts against it; and that in order to conserve the patient's best interests, early and frequent bacteriologic examinations should be made of all discharges in suppurative otitis media, in order to be in position to take advantage of every point that could be of value in determining the necessity for operative procedure; and, further, that having made an attempt to stop its destructive course by operation as soon as there is any indication of mastoid involvement, there is still an uncertainty of prognosis to consider, even after the wound is healed and the patient apparently is well."

Smith<sup>23</sup> concludes:

"The symptoms are very apt to be masked, even in the presence of an extensive destruction of the mastoid process. One of the chief dangers is that of not recognizing the process of the infection. Practically all the cases come to operation."

Page<sup>24</sup> states

"that it is not safe, as a rule, to watch these smouldering cases with a streptococcus mucosus infection longer than two weeks. He had never regretted operating on such cases."

Snee,<sup>26</sup> on the basis of an otologic study of the streptococcus organism, speaks radically as to operation:

"Once the diagnosis has been made operation should not be delayed. The apparent healthy cortical bone of the mastoid and zygoma should not be a deterrent from proceeding to the deeper structures. If your diagnosis is correct you will find the spongy bone and the pneumatic cells involved and filled with mucoid exudate and vascular granulations, removal of which is attended by free hemorrhage."

and closes his article with:

"Parenthetically let me insist that the otologist who does not type every suppurative otitis media, whether acute or chronic, has not done his full duty by his patient or to himself."

Dunlap<sup>27</sup> makes the same arraignment against the streptococcus hemolytic, a strain of which was present in an epidemic of sore throat and mastoiditis.

As to the pneumococcus group, since they were not typed, one cannot tell how many of them have been type III. Its destructive action needs no further mention.

The discussion to this time has included various etiologic bases for the explanation of this syndrome, all of which depend in the last analysis on pressure in some form.

One other theory has been given by a number of observers, and that is an infectious or toxic neuritis. This theory is soundly defended by Knick,<sup>28</sup> who, after carefully reporting five cases, ascribes one to serous meningitis and the others to a toxic neuritis, as follows:

"In conclusion, one may say, among five cases of abducens paralysis in acute otitis media, with or without mastoiditis, a meningitis could be proven but once through an exact examination of the cerebrospinal fluid, and be accepted as the eventual cause of a sixth nerve palsy. In the other four cases the cerebrospinal fluid was negative and there is no authority for the assumption of a serous meningitis. The meningitic origin of the sixth palsy, which by different authorities is given first place, could not be proven in the majority of cases, and the hypothesis of an extradural injury of the nerve by inflammatory affection at the point of the apex of the pyramid could not be denied nor made improbable; rather the negative condition of the spinal fluid and the absence of meningitic symptoms also permit the assumption of an extradural toxic infectious injury, and mainly from the deep perilabyrinth cells to

the apex of the pyramid. I do not pretend to deny the occurrence of meningitic palsy of the sixth nerve, since I myself have seen such a case. But this etiology cannot be proved in a majority of cases, and for that reason possibility of an extradural toxic injury must also be considered, even though the only safe proof, the histologic one, is still lacking."

Alt has been quoted frequently on his varieties of otogenic paralyses based upon an etiologic basis.

1. Reflex paralysis by the route of the vestibular nerve. This will not be discussed, but seems untenable.

2. Paralysis depending upon infective neuritis. That some of the cases herein tabulated may have been due to this is possible; there seems no possible way to controvert such a diagnosis without autopsy and histologic study. But, as Fremel says, "it is such an easy diagnosis to make."

3. Extension of the purulent inflammation from the middle ear in the venous channels of the carotic canal to the cavernous sinus.

4. Deep seated disease of the petrous bone at the apex with localized meningitis. This theory seems to be undoubtedly active in a number of the cases reported.

5. Otogenic diffuse meningitis: whether protective or suppurative is not mentioned. Association with a serous or protective meningitis was rather frequent in this series. Of the fatal cases a final suppurative meningitis was present in fifteen.

6. Extradural abscess. A group of these cases seems to have direct relation to the extradural abscess present.

Pathology of the petrous bone in otogenic paralysis of the abducens, as observed by careful autopsy, is slight in quantity, covering all autopsy records available. However, the material at hand is not too convincing; to offset the abscess cavities at the petrous tip there are others to match them in which no paralysis existed. One case of isolated thrombosis of the superior petrosal sinus was recorded in 172 cases as the probable cause. Shemely<sup>31</sup> reports an otogenic isolated thrombosis of the same vessel without sixth nerve palsy.

It was present in twenty-seven cases of sinus thrombosis, yet in the large majority of sinus thromboses it is not present. Extradural, cerebral and cerebellar abscesses associate with it in this series, but much more frequently it does not appear,



so that other factors must be considered than the mere facts stated above.

One must remember, in dealing with otogenic infection, as with all others, that there are two important factors to be considered, both of which are subject to wide variations in intensity; first, the resistance of the patient toward infection; second, the virulence of the infective agent. The variations in amount and intensity of these two factors may be responsible, with the others mentioned, for the number of cases escaping this paralysis under apparently the same general clinical symptoms.

There are so many points to be considered in a discussion of this syndrome that many of them must go without mention, viz.: associated paralyses of other intracranial and especially of the oculoorbital, nerves; the cerebrospinal fluid; what influence changes in its character from a moderate increase in cell count to coagulation; from a clear, nontoxic fluid to one decidedly toxic; how this may be studied by animal experimentation; further, its influence by changes of pressure; a more detailed study of the various organisms involved, with the symptomatology at various stages; the help to be derived by X-ray studies; a full discussion of the surgery involved; a review of the theories and conclusions as to causation advanced by representative otologists, etc., the attitude of the profession for and against the Gradenigo syndrome as a clinical entity; the limited association of the suppurative labyrinthitis; material of which is at hand, but cannot be utilized.

#### CONCLUSIONS.

1. Isolated paralysis of the abducens associated with irritation of the Gasserian ganglion is a relatively rare complication of acute suppurative otitis media, though less rare than is apparent, for many cases have appeared in the literature under other titles, others are seen and diagnosed, but not recorded, while some are met and not recognized.

2. Pain of Gasserian type is commonly associated with otogenic paralysis of the abducens, so that the Gradenigo triad of symptoms will be present as a rule in any such palsy.

3. Many cases reported under the Gradenigo syndrome are classified incorrectly from an etiologic basis.

4. The thorough study of the infecting organism in any acute otitis, routinely culturing and typing the same, may give indication for operative interference before a palsy occurs, and lessen the mortality following its occurrence.

5. The subject is of sufficient importance to be thoroughly incorporated in every textbook on otology.

6. The mortality of 18 per cent indicates a graver prognosis than has been associated.

#### HYPOTHESES SUGGESTED FOR DISCUSSION.

1. The great variations in size of Dorello's canal as a factor in the occurrence of or the escape from paralysis of the abducens.

2. The asymmetry in size of Dorello's canal on the two sides of the same patient as influencing the occurrence of a contralateral palsy.

3. The development of increased resistance of the nerve on the side of the affected ear in thrombophlebitis or thrombosis of the associated sinus as aiding in determining a contralateral sixth paresis.

4. Anomalous variations in distribution of branches of the basilar artery in relation to the abducentes as influencing compression in meningitis serosa, or cerebral edema, in the production of sixth nerve paralysis of the opposite side. (a) Artery superficial to the nerve on one side, nerve superficial to the artery on the other side; (b) artery superficial to the nerve on one side, nerve free from arterial relation on the other side; (c) artery superficial to the nerve on one side, artery piercing the nerve on the other side.

In (a) and (b) arterial compression or constriction affecting one nerve and not the other; in (c) constricting the nerve on one side and part of the nerve only on the other. In either case, should the arterial compression occur on the side opposite the affected ear, a contralateral paralysis could result.

#### CASE REPORTS.

Case 1.—November 29, 1907, the writer was called to see T. F. B., white, male, age 30, who gave a history of very severe pain affecting the left side of the head for the four preceding days. Temperature 99.3°, pulse 90, respiration 20. Prior to the onset of the pain, which was agonizing at times, he had suffered from a slight head cold. The cold, however, had cleared, and at this time gave no symptoms. It was noteworthy

that at no time had there been any pain whatsoever in the ear, and, although impairment of hearing was marked, the patient had not noted it until his attention was called by examination. Hearing was reduced to C. V. at ten feet, Weber to the left; Schwabach lengthened, and Rinne negative.

Inspection showed an approximately normal external canal. The tympanic membrane, however, was dull, thickened, and showed marked bulging. Incision of the membrane was followed by free purulent discharge. The pain was relieved by the flow of pus, which continued for three days, when pain again became severe, coincident with a decrease in the quantity of purulent discharge. A second incision of the tympanic membrane became necessary twelve hours later. Again, for three days the purulent flow was quite free, and the patient was free of pain. This was followed by a considerable decrease in the amount of pus and an immediate increase of pain. The membrane was again incised, with an increased flow of pus and similar easing of pain. At this time there was some mastoid tenderness, which gradually decreased, and passed away in several days. Temperature during this period had varied from 99 to 100.

The purulent discharge was very free for another week, and then began to gradually lessen, but without return of pain. On December 17th, the patient complained of double vision. Examination showed homonymous diplopia, the separation of images increased on looking to the left—a paresis of the left externus; T. P. R. at this time normal.

The next morning the paralysis was complete. This was shown by a marked convergent squint, accompanied by annoying diplopia and vertigo. The vertigo was promptly relieved by closure of the affected eye, thus indicating that it was due to diplopia and muscle paralysis, and not to an intracranial complication. Further, the left eye could not be rotated externally beyond the midline. An added complication, occurring simultaneously with the complete palsy of the muscle, was a polyarthritides, accompanied by a rise in temperature.

Examination of Eyes. Movement of the lids of both eyes normal; considerable injection of the conjunctiva of the left eyeball. Pupil of each eye, 2.5 mm., reacted freely and equally to L. and C. The media were clear, discs clearly outlined, and

no evidence of papilledema or gross lesion of the fundi. V. 20/15-0°.

Ocular movements were unaffected except in the field of the left externus. Pain was elicited by pressure over the area of the affected muscle and by ocular movement, especially by strong effort at rotation toward the affected side. Bacteriologic examination at this time showed streptococci and staphylococci, streptococci predominating. No lumbar puncture was done or X-ray study made at this time. A skiagram taken recently revealed a pneumatic mastoid of very extensive development.

The purulent discharge ceased within ten days, and the arthritis improved more rapidly than the palsy. By January 18, 1908, no diplopia could be elicited by extended ocular rotation to the left, so that all symptoms of the paralysis had cleared. There has been no recurrence.

Comment: 1. The onset of severe left head pain was the first symptom to be noted. The pain was relieved by drainage of pus and recurred with retention.

2. The abducens palsy appeared three weeks after the onset of the otitis, when the disease was on the wane, and it finally disappeared thirty-two days after its appearance.

3. The complicating polyarthritis arose coincidentally with the palsy, but had healed at least two weeks earlier.

4. There were definite signs of local inflammatory reaction about the affected muscle.

Case 2.—On December 21, 1916, I first saw C. S., white, male, age 26. He was suffering with an acute mastoiditis, complicating a middle ear suppuration. This was the sequence of a severe head cold and sore throat, which had attacked him two and one-half weeks earlier. The tympanic membrane had ruptured spontaneously eight days before without relief to an intense pain in the right temporoparietal region. This pain was so severe that he remained in bed for thirteen days prior to my first visit.

Examination: Anemic looking young man with marked pallor. Facies appeared dull and rather stupid, cerebration slow. Right ear, hearing good, otoscopic examination negative. Left ear, profuse discharge from external auditory meatus, canal much narrowed, posterior superior wall broken down, other

landmarks obscured. Spontaneous perforation of superior posterior quadrant of tympanic membrane, which had a nipple like protrusion. No external swelling over mastoid, but pain elicited by pressure at the cardinal points.

Immediate operation was advised but not agreed to. Five days later, December 26th, the patient was removed to the Blair Hospital, and a simple but thorough mastoidectomy performed the following day. His general condition at this time was decidedly worse. Admission temperature  $97.3^{\circ}$ , pulse 60, respiration 20, cerebation slow, somewhat stuporous, but moaning at times from severe head pain.

Operation: Periosteum adherent, cortex thick and vascular, very large celled pneumatic mastoid, cells extending well into zygoma, part of mastoid completely broken down and a large perisinus abscess present. Pus welled up under pressure. A few granulations appeared on the sinus where it was exposed to view by necrosis of the inner plate. The sinus itself seemed to be functioning. On removal of tegmen antri the dura seemed normal, although not pulsating.

The antrum was large and filled with pus, as were the cells everywhere. The opening to the tympanum from the antrum seemed free. The tympanic membrane was incised.

Laboratory Findings: Urine relatively negative throughout his illness. Bacteriology—pus from the mastoid mixed infection, streptococci, staphylococci and diphtheroids.

Blood: Red cells, 4,500,000; white cells, 16,500, polys 90 per cent. Wassermann negative. X-ray not taken.

Lumbar Puncture: Cerebrospinal fluid under pressure—low cell count—no bacteria found. Wassermann negative.

Seven days after the operation a paresis of the right abducens was noted; this two days later became a complete paralysis. The paralysis cleared gradually and disappeared within three weeks of its onset. At the same time weakness of the seventh nerve on the same side was noted, which likewise passed with the clearing of the abducens. For a few days following the operation the patient was inclined to be stupid, then for two or three days talkative and somewhat irrational, later much depressed, but after two weeks his normal mentality came into evidence again. His temperature had a subnormal tendency throughout his stay in the hospital.

Eyes: There were no disturbances of the intrinsic or extrinsic ocular muscles, except the paralysis of the right externus. On admission to the hospital there was a low grade papillitis in both eyes, which remained stationary for a couple of weeks after the operation, and then gradually passed away. Vision was not taken on admission, but two weeks after the operation it was 20/20 0°.

After operation, the head pain which affected the right temporoparietal, and, at times, the right frontal region and eye, and occasionally the left eye, was not relieved. It continued of the same severe type for one week, less severe for another week, and occasionally severe after the third week. It was entirely gone by February 1st, when the patient was discharged from the hospital. There was very free purulent secretion from both the mastoid wound and the external auditory meatus for three weeks following the operation. This gradually lessened, and on the date of discharge both middle ear and mastoid were dry.

Six weeks after he left the hospital an attack of violent pain affected the right ear and head. Incision of a bulging tympanic membrane brought a profuse flow of pus, which persisted for three weeks; the first week the discharge was extremely free, then it gradually lessened, and finally cleared. The pain passed away with the cessation of the free purulent discharge and did not recur, nor was there any symptom of weakness in the external rectus. Three weeks after the ear was dry hearing was again normal. There has been no recurrence.

Note: In this case the very free discharge of pus by way of the external auditory canal, when the exit from the tympanic cavity to the antrum and mastoid wound seemed free, was noteworthy. In fact, the purulent flow was so large that it could not possibly have originated in the tympanic cavity alone.

Case 3.—Charles H., white, age 19, was brought to my office April 12, 1923, complaining of severe pain in left side of head and face, and great bodily weakness. He gave a history of a sudden attack of agonizing pain in the right ear, commencing about fifty-six hours before. Twelve hours after the initial attack of pain, spontaneous rupture of the tympanic membrane occurred, with free catarrhal discharge. The pain ceased for

twelve hours, then was moderate in character for another twelve hours, but for the last twenty-four hours had been extremely severe over the left side of the head and face. Nauseated, constipated, no appetite.

Previous medical and ear history was negative until seven or eight months ago, when the patient was affected by a severe head cold and an irritating chest cough. The symptoms were severe for several days, then gradually ameliorated until, about three weeks after the onset, recovery had ensued. At this time a similar attack developed with the same symptoms and course. These attacks followed one after the other, until recently. One week ago, following exposure to wet and cold, the patient developed a head cold with coincident swelling over the mastoid. This coryza had cleared by Tuesday, April 10th, the date of onset of pain in his ear.

Examination: Temperature 100.3,° pulse 90, respiration 20. Fairly nourished male, looks ill and depressed.

Right Ear: Hearing normal; otoscopic examination negative. Left ear: Auricle pushed forward. Moderate swelling over midhalf of mastoid, pain on traction of the auricle, and marked pain on pressure over the cardinal points of the mastoid. A suggestion of fluctuation on palpitation over the swelling in the antral region. Ear was filled with débris. After cleansing, external canal seemed to be narrowed, postsuperior wall breaking down, a free catarrhal discharge through a spontaneous perforation in lower posterior quadrant of the tympanic membrane. The landmarks were all obscured.

Nose: Septum deflected to left, nasal mucosa congested, turbinates slightly swollen, no purulent secretion seen. Negative pressure induced a slight flow of serum but no pus.

Mouth: Teeth in good condition; tonsils submerged and contained caseous débris; pharynx markedly inflamed. Chest seemed negative. Postcervical glands enlarged.

Hearing: Right ear, conversational voice, 40 feet; whisper, 20 feet. Left ear, loud voice at six inches.

Eyes: Vision 20/20 0°, ocular rotations normal. Spontaneous nystagmus in all directions of movement of the eye, with a very marked combined horizontal and rotary nystagmus to the left on ocular rotation to the left. Pupil 2.5 mm. 0° irides

respond to light and convergence and consensually. Fundi negative, no evidence of pressure.

Patient admitted to Blair Hospital, April 12, 1923, at 8 p. m., where the following laboratory and roentgen ray studies were made:

Urinalysis: Sp. gr. 1021; albumin, faint trace, no sugar, acetone or diacetic acid. Microscopic, no casts, but many leucocytes and epithelial cells.

Blood: Red cells, 5,210,000, white cells, 16,950, polymorpho, 70 per cent. Wassermann negative.

Spinal fluid: April 15th, pressure 6 mm. Hg., water clear, cell count, average one cell to field. Globulin, Pandy, slight reaction, Nonne negative. Wassermann negative.

X-ray report: Small celled mastoid, hazy throughout, sinus well back. Nasal accessory sinuses negative; transillumination of sinuses, negative.

Operation: The following morning a simple mastoidectomy was done on the basis of the above findings. The tissues covering the mastoid proved to be but moderately infiltrated; the periosteum very adherent to the bone, which, when exposed, bled freely from many points. The mastoid proved to be small celled, and in the congestive stage, so that extreme hemorrhage complicated the operation. The antrum was small and not easily located, and edematous tissue filled the cells in the antral region. No free pus was found in the mastoid except a small amount in the antrum, which evidently came from the tympanum. The tympanic membrane was freely incised.

Course of disease following operation: Temperature went to 100.6 the evening of the day of the operation, then hovered about 99 for the next six days, when it dropped to subnormal on the 19th, the day on which paresis of the sixth became evident. After this it varied from 97 to 98 for the ensuing eight days. On the 27th it rose to normal following the removal of 35 c. c. of spinal fluid, where it remained until the 29th, when it again became subnormal for a period of four days, after which it returned to about normal.

Pulse varied from 60 to 90, but was generally between 70 and 80. Respiration averaged 20 for the entire illness.

Pain: The pain was agonizing. It affected the side of the head and face, and the upper and lower jaws. Later it affected



the left eye and the supraorbital region. From April 11th to May 11th, inclusive, the pain was persistent, affecting the distribution of the fifth. It was relieved by narcotics, anodynes or opiates for a short time only. At no time was there complete relief for six hours consecutively, until May 10th, when it subsided rapidly.

Gastrointestinal: Nausea and vomiting were present every day during the entire time the pain persisted. Constipation was marked, appetite poor, and he was unable to retain food part of the time.

Tympanic cavity and mastoid: First dressing was done two days after mastoidectomy, on account of the intense pain on the operated side of the head. There was no pus from the mastoid, but very free purulent discharge from the middle ear through the external canal. Four days later pus came from the mastoid wound, but less abundantly than from the canal. On April 27th the patient was taken to the operating room, and, under ether, search was made for cells not uncovered originally. Some were found in the antral region, and the bony wound was extended in all directions. The sinus plate was sound and the tegmen antri unaffected; no necrotic bone found anywhere, but granulation tissue was present in all cells uncovered; part of the tip left at former operation on account of the severe hemorrhage was also removed. The opening from the aditus to the antrum was freely curetted. Following this, free drainage of pus continued from both the external canal and the mastoid wound. May 5th, twenty-four days after the first operation, pus was still draining freely from the external canal, although beginning to lessen from the mastoid cavity.

A week later the external canal flow had lessened materially, and on May 16th, when the patient was discharged from the hospital, the middle ear was almost dry, and after another week all secretion had ceased and has not recurred. The closure of the mastoid wound by granulation was uneventful.

The eyes: On April 20th, eight days after admission to the hospital, a paresis of the left external rectus appeared. This increased slowly for a week, when, on April 27th, the paralysis became absolute. It was evidenced objectively by a convergent squint, and by inability to rotate the eye to the left beyond

the median line. Subjectively, by diplopia homonymous in character, the distance between the images increased as the eyes were rotated to the left. Twenty-four hours later, following the removal of 35 c. c. of cerebrospinal fluid, the absolute character of the paralysis had disappeared, and the eye could be rotated  $20^{\circ}$  to the left of the median line. From that time for ten days the paresis varied in extent, but by May 16th there was considerable improvement, and on June 11th there was no sign of palsy except by extended rotation to the left, when diplopia could be induced. The intrinsic eye muscles were unaffected at any time. The eyegrounds were negative throughout. The edge of the disc was clear cut, the physiologic cup and lamina cribrosa easily seen.

Laboratory: Urine relatively negative throughout. Blood: White blood cells varied from 16,950 on admission to 8,550 four weeks later, polymorphs running from 70 to 80 per cent. Blood culture, May 3rd, negative. Blood, Wassermann test, negative.

Spinal fluid: Specimen removed April 15th under normal pressure. April 27th, 35 c. c. removed under high pressure; April 30th, under moderate pressure; May 3rd, slight pressure; May 9th, normal pressure. The fluid was water clear and contained but few cells and no bacteria at any examination. Wassermann negative.

Pus from middle ear, pure pneumococcus. Sputum, pneumococci, streptococci and staphylococci. No T. B.

Interesting Points.—1. The sudden onset of pain, followed by spontaneous rupture of the tympanic membrane twelve hours later; the persistence and severity of the pain which remained practically the same during the entire course of the pus discharge, and which remitted when it became less free.

2. The onset of the abducens paresis coincident with that of a subnormal temperature; the absolute paralysis, lessening somewhat and the temperature returning to normal simultaneously following the removal of a large quantity of cerebrospinal fluid under high pressure.

3. The continued free purulent flow from the middle ear via the external auditory canal, after the mastoid and antrum had been opened, and, especially, after a secondary enlargement of the cavity with especial attention to the opening of the adi-

tus into the antrum. This is the more noteworthy in face of the small celled type of the temporal bone; for purulent discharge continued for approximately six weeks after the spontaneous rupture of the tympanic membrane.

Examination of this patient November 11, 1923. Hearing in left ear returned to practically normal. Ocular muscles, an esophoria of 2° at 20 feet by Maddox rod test.

Case 4 (Courtesy of Dr. M. S. Ersner).—S. H., age 8. Acute mastoiditis (right side). February 14, 1924. Patient was admitted to hospital on February 14, 1924, with family history negative, and with a previous history of having had measles and mumps. The present condition started with a cold four weeks ago, which was followed by ache in right ear and a spontaneous rupture of drum. The left ear drum was incised later. One week later the right ear drum was incised, but the condition did not clear, and discharge and pain behind the ear persisted.

Examination.—1. Profuse aural discharge.

2. Impairment of hearing. W. V. 6 feet; watch 6 inches; S. V. 20 feet; Weber right; Schwabach plus; Rinne negative; loss of low tones, but high and middle heard, but less duration.

3. Postauricular edema, folds thickened and somewhat obliterated.

4. Loss of mobility of tissues over mastoid (periostitis), auricle protruding slightly.

5. Urine: February 14, 1915, trace albumin; acid; finely granular and hyalin casts.

6. X-ray: General blurred haziness and thickening of the cell walls throughout mastoid process of both sides. On the right side, in the posterior zone, and extending toward along the inferior cells, well toward the true mastoid tip, there is evidence of breaking down of the cell walls. I believe this is bone necrosis. The left side shows a similar disturbance about the same area, but to a less degree. Here there seems to be some evidence of bone repair. The process here does not seem to be as marked or as active as on the right side. Eidman.

Operation: February 14th. Ether anesthesia. Modified blood clot closure.

(1) Periosteum stripped easily. (2) Cortex very thick, but not congested. (3) Cells congested and necrosis at the tip.

(4) Large cell posterior to lateral sinus filled with pus. (5) Softening of the posterior canal wall. (6) Deeper layers of cells over the sinus plate involved. (7) Culture of pus from the mastoid cells: *Streptococcus*.

• Postoperative period: February 15, 1924. Postoperative rise of temperature ( $103.3^{\circ}$ ). The next day the wound was dressed and some of the stitches removed to provide for drainage, as there was a straw colored serum coming from the wound, indicating breaking down. From this time on the wound discharge was very free. On February 22nd the wound was still wide open and draining, but no discharge from the ear and the temperature was normal. On the 25th the wound was granulating well and closing up with no discharge from the ear, but drum was still red and no landmarks visible.

The patient was discharged on the 26th with temperature normal and the wound well on the way to closing up.

S. H., age 6. Secondary mastoiditis. March 20, 1924. Patient reentered the hospital on March 20, 1924. A simple mastoidectomy was done on the right side on February 14th. The present condition started just one month later (March 15th) with nausea, vomiting and headache, and some sensitiveness to light. On March 19th the patient complained of double vision, and the next day it was noticed that both eyes turned inward, but the right eye more than the left.

Examination.—1. Temperature  $101^{\circ}$ . Headache and slight rigidity of the neck.

2. External wound had ceased to discharge and ear was dry.

3. Some tenderness over the mastoid region.

4. Urine: Acid, 1012; slight trace of albumin; 20-30 leucocytes; epithelial cells.

5. Blood: Leucocytes 11,925; 15 small lymph.; 8 large lymph.; 77 polym.

6. Eyes: Pupils nearly equal. O. D. a shade larger. Paresis of left external rectus. O<sup>2</sup> decided papillitis.

7. Physical: General condition fair. Heart and lungs negative. No special signs. Kernig, Babinski, contralateral, and knee jerks all negative. Left external rectus paresis present. Following day: Condition remains the same. All special signs are negative. Slight neck rigidity noticed. No diplopia. Mental

state good. External rectus paralysis somewhat better. No evidence of meningitis.

8. Eyes: March 22nd, 2 p. m. Still decided papillitis, veins very much engorged. Right nervehead a little more prominent than left. Outward excursion of left more limited than right. Evidence of intracranial pressure. After various consultations, Dr. Ersner decided to explore the field of previous operation.

Operation: March 22, 1924, 4 p. m. N<sub>2</sub>O and O. Operation took one hour.

Findings: (1) Excision and removal of scar. (2) Exposure of the middle fossa. Dura looked normal, no tension present. (3) Roof of mastoid found to be of hard healthy bone. (4) Exposure of the lateral sinus down to jugular bulb. Bony wall over the sinus found soft and covered with edematous mucous membrane with occasional suspicious pus droplets. Under portion of lateral sinus was covered with soft tissue and edematous mucous membrane. The sinus pulsated and was not boggy. The sinus wall was congested and was of a reddish tint and covered with a thin layer of fibrin.

Postoperative period: March 22nd, 10 p. m. Temperature reached 104°, and by 3 p. m. came down to 100°.

March 23rd, 4 p. m. Temperature went to 97.2°. Pulse 102.

March 24th. The wound was dressed. The lateral sinus looked well. Wound good. Little change in the fundus. Paresis of external rectus less marked.

March 25th. No neurologic findings with the exception that the left leg shows some asynergia as compared with similar movements of right leg. All reflexes present and normal. No meningitic symptoms. The wound was dressed and found in good condition. Temperature 101°.

March 26th. Still marked swelling of papilla and engorgement of veins. Both more marked in right than left. Urine showed a trace of albumin with few leucocytes. Wound was dressed and found in good condition. The white cells were 14,950; 18 small lymph; 5 large lymph; 10 transitionals; 65 poly; urine showed a faint trace of albumin. Temperature went from normal to 102°.

March 27th. Wound was dressed and found in good condition with evidence of granulations. Temperature again came down to normal.

March 28th to 30th. Daily dressings. Urine showed traces of albumin and occasional hyalin casts, leucocytes and squamous epithelial cells. The temperature fluctuated between normal and  $102^{\circ}$ , but on the night of the 30th it went to  $97^{\circ}$ .

March 31st. Marked recession of the papillitis, but still in evidence. General examination discloses no abnormalities. All symptoms subsided, muscle and reflex actions normal. Temperature continued below normal.

April 12th. The signs of papillitis are hazy, but there is no elevation. The vessels are normal in size and course. During the last two weeks the temperature persistently remained at normal or a little below. The wound was dressed less often during the remaining time, as it was in very good condition and healing well. Patient discharged.

Case 5 (Courtesy of Dr. M. S. Ersner).—H. W., age 17 months. Acute bilateral mastoiditis. March 13, 1924. Patient entered hospital on March 13, 1924. Family and previous history negative. Present condition started several weeks ago with pain and discharge from both ears. Mother also noticed simultaneously that one eye turned in.

Examination.—Right: (1) Profuse creamy discharge. (2) Sagging of posterior superior canal wall, assuming nipple like protrusion. (3) Folds more prominent and slightly thickened. Slight protrusion of auricle.

Left: (1) Profuse purulent creamy discharge. (2) Sagging of posterior superior canal wall. (3) Folds slightly lifted with lower fold smoothed out. (4) Urine: Negative. Occasional leucocytes and squamous epithelial cells. (5) Blood: 3,650,000; 10,100; 68 per cent hemog.; 64 small lymph; 35 polym; 1 eosin. (6) X-ray: No appreciable difference between the right and left mastoid processes. Left side looks definitely normal. The semicircular canals clearly visualized. There is a faint suspicion of infection, the posterior cells on the right side. This evidence is not sufficient to be regarded with much weight. (7) Eye: Slight veiling of the edges of the discs. Apparent weakness of the left external rectus muscle, March 13th. March 16th, no evidence of intracranial pressure.

Operation: March 15th. Blood clot closure with drain. Right side. (1) Periosteum stripped easily except at the bottom. (2) Cortex slightly roughened and found thick. (3)

Cavity under the cortex filled with free pus. (4) Involvement of the zygomatic cells. Culture: *Streptococcus*. Left side: (1) Periosteum stripped easily. (2) Cortex moderately thick. (3) Cavity under cortex filled with free pus. Culture: no growth.

Postoperative Period.—March 18th. Removed stitches and inserted gauze drain. Drainage abundant.

March 19th. Wounds draining freely; also considerable discharge from ears.

March 20th. Right wound wide open; left wound coated in upper half. Good drainage.

March 27th. Temperature normal. Wounds granulating and nearly closed, only slight drainage. Both ears dry. Drums assuming normal grayish color. Weakness of external rectus disappeared and no strabismus can now be detected. Discharged to be cared for by family physician.

Case 6 (Courtesy of Dr. H. M. Goddard.)—H. R., female, white, age 7 years. Past medical history P. M. H. An undernourished child with feeding difficulties, subject to repeated colds. Had a year ago an attack of acute otitis, which rapidly healed.

Present illness: April 17, 1924, the patient was taken ill with influenza affecting the upper air passages, and complicated by a light grade pneumonia. April 20th, onset of bilateral otitis media. The right drum was incised on this day and two days later. Discharge purulent and moderately profuse. Left drum redness subsided in a few days. The right ear was dry, and other symptoms had cleared by the 18th day of illness. Two days later, on May 6, 1924, the patient developed otitis media on the left side. The following day the tympanic membrane was incised and purulent discharge followed. Clinical signs of mastoiditis now came into evidence, and on May 9th the patient was removed to the hospital. A simple mastoidectomy was done. Pus under pressure and granulations were found. The temperature remained high ( $103.4^{\circ}$ ) in the evening for seven or eight days, although the mastoid in the ear drained well. At this time the right otitis again became active, and the ear discharged profusely. The patient, however, did not improve, and evening temperature continued. The left ear, twenty-five days after operation, continued to discharge, while a

profuse purulent secretion persisted from the right ear. There were no clinical signs of mastoiditis.

On June 11th, twenty-six days after the right otitis had again become active, a complete paralysis of the right external rectus was noted. There were no other signs of intracranial complications. Leucocyte count, 14,000. A consultation was held two days later, when it was decided there must be some pressure on the right sixth nerve. On June 15th a simple mastoidectomy was done on the right side. Pus was found in small quantity at the tip with granulations and hypertrophic mucous membrane in the other cells. Wound closed with drainage below.

The postoperative course was uneventful, the mastoid wound having healed by July 1st, while the right ear became dry a week later. The sixth nerve paralysis had disappeared by August 8th, or more than eight weeks after its appearance. A record of the pathogenic organism in this case could not be found.

Case 7 (Courtesy of Dr. H. M. Goddard).—C. R., male, white, age 16 years. Chief complaint, pain and loss of hearing in right ear. Past medical history negative, except for influenza during the recent epidemic, and recent night sweats, loss of weight and strength.

History of present illness: August 2, 1924, onset of acute otitis with high fever, the sequence of a sore throat which had healed in one week before. The following day spontaneous discharge occurred, and two days later he noted that he was entirely deaf.

Physical examination, August 14, 1924, on admission to the hospital, negative, except for discharge from the ear. The tympanic membrane showed a red polypoid mass at its posterior superior aspect, a small perforation beneath, canal normal. Nasal examination negative.

August 14th, culture from throat showed pneumococci, staphylococci and streptococci. Culture of pus from right ear showed staphylococcus albus.

August 21st, the discharge had almost ceased. At this time a complete sixth nerve paralysis, associated with a severe peri-orbital and suboccipital neuralgia developed. The eyegrounds



were negative. X-ray of mastoid practically negative. Neurologic examination negative, except for sixth nerve palsy.

August 27th, a simple mastoidectomy was performed, and very little pus found in the antrum and subantral cells. Polypoid mass of the tympanic membrane removed. Following the operation the pain decreased, the ear drained profusely at first, later decreased, but the paralysis remained constant.

On September 3rd, twelve days after the mastoidectomy, the patient had the first symptoms of a fulminating meningitis, which resulted in his death twenty-six hours later. Autopsy was refused. He ran a light evening temperature throughout his illness until meningitis developed. Pulse rate, average 72; respiration 20. Urine was negative throughout. Blood count, red cells 4,000,000, white cells 13,900, polys. 74 per cent.

Case 8 (Courtesy of Dr. B. N. Colver).—L. D., female, age 12. The patient was first seen in consultation October 10, 1923. The following history was given: Has had a cold in her head for several weeks. During summer and fall had been swimming nearly every day. Had a slight earache in the right ear, when the cold first started. Complained of headache recently, especially at night. In past two weeks off and on has been sick in bed. About ten days ago grew much better and was up and about with normal temperature for a couple of days. After this the head began to ache, she complained of nausea, but really vomited only once. The past few days has complained of blurring and dancing of vision. The headache and pain seemed more marked on the right side.

With no facilities in the home for detailed study an immediate diagnosis was made of acute right mastoiditis, evidencing meningeal involvement with Gradenigo's syndrome. The family hesitated about taking immediate steps and brought the patient into the hospital on October 5th.

The fundus examination showed the left fundus negative, the right fundus slight blurring of the borders of papilla. The right sixth nerve was found partially paralyzed.

The X-ray examination showed extensive cloudiness throughout the mastoid area. The blood examination showed red cells 3,660,000; white cells 23,400; Hg. 70 per cent. Blood culture and spinal fluid were taken, but it was felt that imme-

diate surgical interference was justified, even though the meningeal involvement was evident.

On the afternoon of October 5th a complete mastoid exenteration (right) was performed. The cells were filled with a gummy pus; there was no dural exposure. For the first 24 hours the course seemed to be favorable, but the temperature, which had been moderately high, began to mount, and the progress of diffuse meningitis became apparent.

The bacterial examination of the mastoid pus showed few pneumococci and few hemolytic streptococci; the blood culture developed many hemolytic streptococci; the spinal fluid developed hemolytic streptococci and a cell count of 500 cells per c. c.

On October 6th, the white cell count was 22,200; by the 8th it had sunk to 9,000. The patient succumbed on October 10th; necropsy refused.

The points in this case are: 1. The insidious nature of the ear symptoms. 2. The lack of recognition, by those in attendance, of this early sign of extension. 3. The question of when the meningeal irritation became true meningitis. 4. The question of whether it is justifiable to take the small chance of immediate surgery or to accept the almost inevitable fatal outcome, without any effort to stem the tide.

Case 9 (Courtesy of Dr. Ivor Clark).—October, 1919. Mrs. C., age 35. History: No prior disease of ears. Ten days before admission there was sudden acute pain in the ear and side of head; the tympanic membrane had been incised on the fourth day; a moderate flow of pus ensued, but the head pain was not relieved. Temperature of moderate degree. Examination of the ear drum showed the incision to be open and a moderate drainage of pus.

There were no well marked clinical symptoms of mastoiditis, but on account of the agonizing pain, a simple mastoidectomy was performed the following day. The mastoid proved to be of the small celled pneumatic type, there was very little free pus, some bone softening and the cells filled with granulation tissue. The pain was not relieved by the operation.

A septic temperature with associated symptoms of sinus thrombosis now appeared. This diagnosis and operation advised. Operation was refused at the time, but consented to

two weeks after the mastoidectomy, when induration over the internal jugular vein appeared and the patient's general condition became alarming to the family.

Second operation: The lateral sinus was exposed, the wall of the sinus discolored, the sinus itself doughy and not functioning. The internal jugular vein was ligated and resected; owing to the serious condition of the patient, nothing further was done.

Following the operation, pain ceased, temperature subsided and the patient did well for ten days, when again a septic temperature developed. Antistreptococci serum was injected intravenously with good results. Five days later a complete paralysis of the sixth nerve developed on the opposite side. This passed away in six weeks. Her general recovery was slow but uneventful.

Case 10 (Courtesy of Dr. Ivor Clark).—January, 1922. Girl, 17 years of age. History of an acute otitis in childhood, which healed promptly without complications. Following a head cold, an acute suppuration of the right ear arose; a spontaneous discharge of pus occurred five days before. On admission to the hospital temperature was  $101^{\circ}$ ; there was a profuse purulent discharge from the right ear and an intense pain affected the right side of head. Two days later a simple mastoidectomy was done; there was extensive necrosis and free pus in abundance.

For the ensuing week, the patient's pain was relieved and her general condition good. Now a septic temperature appeared, associated with very severe head pain on the right side, and gradually rigidity of the neck and tenderness over the internal jugular vein of the right side developed for one week.

Second operation: The internal jugular vein was ligated and resected. Pain and temperature were immediately much improved and the patient did well until the tenth day, when a paresis of the sixth nerve of the opposite side appeared. This developed in five or six days to a complete paralysis. The paralysis eight weeks later was still complete. At this time the patient had made an otherwise complete recovery.

Case 11 (Courtesy of Dr. N. S. Weinberger).—J. H., male, white, age 36, presented himself for examination on January 5, 1923.

History: Left ear began discharging three months ago during a cold in the head. Has had no pain; ears runs continuously. Past two days felt sick and has pain over left side of head and face. Jaw feels stiff.

Examination: A. S.—Free discharge of mucopus. Posterior superior canal wall slightly bulging, discharge pulsating. No tenderness over mastoid. Perforation  $1 \times 2$  mm. up and back. Advised immediate admission to hospital—refused.

January 7th, patient returns with excruciating pain in head, vertigo, nausea, vomiting. Admitted to hospital. Morph. sulph. gr.  $1/6$  every four hours gave some relief. White blood count 12,800, differential 84-16.

January 8th, simple mastoid operation. Extensive necrosis, especially involving the dural plate above, which was removed.

January 9th, spinal fluid negative.

January 12th, report on blood culture, streptococcus mucosus capsulatus. No X-ray picture taken.

January 13th, pain in head again severe, irrational at times. Diplopia, vertigo and intense photophobia. Paralysis of left abducens complete.

January 20th, blood culture negative. Incision and drainage abscess right forearm.

January 22nd, incision and drainage abscess left forearm. Blood transfusion.

January 25th, incision and drainage abscess left leg.

February 13th, incision and drainage abscess neck.

February 17th, exploration of lateral sinus for thrombosis. None found. Vein not disturbed.

Brain abscess was suspected from the beginning, but no operative search was made for it. Eyegrounds were negative. Headache, although intense off and on, and accompanied by irrational periods, persisted until about three weeks after operation. Improvement was gradual, however, and the patient was discharged from the hospital March 17, 1923. The abducens paralysis had not fully recovered until about a month after discharge from hospital. Hearing returned to whispered voice at ten feet. Temperature range was 97 to 104.2. Septic course up to January 26, 1923, and normal thereafter. Recovery was complete with no further trouble. The perforation in the drum remains but the ear is dry.

Case 12 (Courtesy of Dr. W. A. Krieger).—November 23, 1922. B. M. B., girl, age 11 years. History of coryza for one week; spontaneous rupture of drum and profuse discharge occurred twenty-four hours after onset of otitis; large perforation in posterior inferior quadrant.

Three days after onset of otitis the patient was operated upon (November 26, 1922) for acute mastoiditis, right. The entire group of cells were found involved. Simple mastoid operation was done. Culture from wound showed short chain streptococcus. Temperature dropped by lysis from 103° to normal on fourth day. At the same time discharge from the external canal ceased.

On the eleventh day after the operation the patient complained of severe headache, and on the fourteenth day developed paralysis of the right external rectus. On account of this complication the mastoid wound was opened and kept irrigated with Dakin's solution every three hours. After the sixteenth day there was no complaint of headache. During this time there was no rise in temperature.

The patient was discharged from the hospital on December 24, 1922, the twenty-eighth day after the operation. The wound was entirely healed and paralysis greatly improved.

The patient was seen at intervals until February 6, 1923, at which time the paralysis was very slight.

Case 13 (Courtesy of Dr. Edwin Cobb).—Mrs. H., age 50 years, came for treatment of a left sixth nerve paresis, which had arisen about one month before the course of a chronic suppurative otitis media in the left ear. She was able to hear conversational voice at one foot. Labyrinth was functioning. There was a large perforation in the tympanic membrane, with a very large quantity of dark pus in evidence. Right ear: hearing conversational voice at 30 feet. Inner ear: active by caloric tests. Cultures from left ear showed a mixed infection.

A radical mastoidectomy was done with complete removal of all diseased structure. Recovery from the operation was uneventful, although the paralysis persisted for some weeks, when it gradually cleared. There has been no return.

Case 14 (Courtesy of Dr. John J. Sullivan, Jr.).—Adult, male, white. The patient had undergone a primary and sec-

ondary mastoid operation for relief of an otitic suppuration from which he suffered.

At first examination his temperature was normal and remained so during the entire period of his hospital stay. There was a fairly profuse discharge from the middle ear, but a small portion of the tympanic membrane remained. The patient was advised to undergo a radical mastoidectomy, to which he agreed. After an extensive exenteration of diseased bone in all directions, I noticed a small amount of pus behind the external semicircular canal. On probing at this point an opening was found leading to a cavity which extended for three-quarters of an inch toward the apex of the petrous, from which a considerable quantity of pus came freely. Toward the close of the operation the patient became cyanotic, and an acute bilateral exophthalmos developed, and at the same time a marked convergent squint. The wound was left open and a small drain inserted in this deep petrosal cavity. A very severe temporal head and facial pain, from which he had suffered for some time before his admission to the hospital, disappeared within forty-eight hours. Five days after the operation a facial paralysis of the same side ensued. The purulent discharge persisted for one month after the operation. The exophthalmos disappeared in the course of a week. The external rectus paralysis also passed away about the same time. The facial palsy gradually improved. The patient made a good recovery.

Case 15 (Courtesy of Dr. Joseph C. Beck).—Mrs. T. W. complained of very severe pain in the side of face and head, and also of lagging of face and overflow of tears on that side. She gave a history of a chronic suppuration in the left ear. History otherwise negative, and lues was positively excluded.

Examination of the ear showed a margin perforation of the tympanic membrane posteriorly and inferiorly, with a thin layer of granulations, from which was discharging a foul smelling pus. The hearing for voice was negligible, and with the noise apparatus at the right ear the left ear showed complete deafness. Hot and cold water caloric tests brought no vestibular reaction. To turning test there was a slight after nystagmus, and especially from the vertical canals. There was no evidence whatever of intracranial disease. Spinal puncture was made and gave no evidence of pressure. The examination

of the fluid was normal. This procedure, however, appeared to relieve the patient of much of the pain for a time. There was a positive facial and sixth nerve palsy present. The pain, which was intense, was distributed over the entire region of the face, top of the head, and also into the throat. A radical mastoidectomy was performed, which showed the typical sclerotic mastoid with fistula leading toward the perforation. The patient recovered completely, including the return of a fair amount of hearing.

Case 16 (Courtesy of Dr. N. S. Weinberger).—This patient was seen in consultation in April, 1923. There had been abortion, with retained placenta and uterine infection. At that time a septic temperature, with chills and fever, and a foul lochia, were in evidence.

The patient gave a history of the onset of an acute suppurative otitis media ten days before, followed by a spontaneous discharge the following day, and four days later the discharge suddenly ceased. On the same day there was a complete paralysis of the left abducens. There was intense pain in her head on the side of the palsy. Blood culture was negative, as was the Wassermann reaction. Spinal fluid was not taken, nor was an X-ray of the mastoid. Examination of the ear showed a reddened drum without bulging, with tenderness to pressure over the mastoid. An ice bag was applied to the mastoid, but no medical or surgical treatment of any kind was done for the ear or mastoid. Hearing returned to normal and mastoid symptoms disappeared within two weeks. Her uterine condition cleared in two months as she became entirely well, excepting for the sixth nerve paralysis, which persisted for three months and then cleared entirely.

Case 17 (Courtesy of Dr. George E. Shambaugh).—A nurse developed an acute suppurative otitis media, associated with an intense head pain on the same side. Incision of the tympanic membrane on the tenth day brought a moderate discharge of pus. The pain, however, was not relieved. An X-ray picture of the mastoid showed the cells to be clouded. There were no clinical symptoms of mastoiditis. Because of the intensely severe pain, which had been continuous for two weeks after incision, a simple mastoidectomy was done. The cells were full of granulation tissue, and very little free pus was

found in the antrum. The mastoid was of the small celled pneumatic type. A culture from the mastoid pus showed the streptococcus. The mastoid wound healed and the ear became dry promptly, but the intense pain continued. Her temperature during this entire period had been but little above normal, rarely reaching 100°.

One month after the simple mastoidectomy a right external paralysis (the same side) developed. A secondary operation was now instituted, and its field was extended into the zygoma and above the petrous, where an extradural abscess was located and evacuated. The severe pain disappeared within a day or two, and the paralysis within a couple of weeks, and the patient made a prompt and thorough recovery.

Case 18 (Courtesy of Dr. N. A. Fisher).—February, 1921. Girl of 15. The patient gave a history of the grippe, which was followed by an acute suppurative otitis media on the left side three weeks before. An early spontaneous rupture of the tympanic membrane, followed by a profuse purulent discharge, was later complicated by a severe pain in the left periorbital and temporal region and a complete paralysis of the left abducens nerve, and these were present on admission to the hospital.

The X-ray picture showed a clouded but pneumatic mastoid. A simple mastoidectomy was done on the second day. The operative findings included granulation and pus in a pneumatic mastoid of large extent.

The culture of pus from the mastoid showed the pneumococcus to be the pathogenic organism.

The postoperative history records immediate relief from pain and an uninterrupted convalescence, while the abducens paralysis cleared in about four weeks.

Case 19 (Courtesy of Dr. R. E. Robinson).—March, 1919. Woman, age 53. She had had no previous ear disease. The patient had developed a head cold a few weeks before, which was followed by an acute suppuration of the right ear. Spontaneous perforation of the tympanic membrane had occurred on the second day, since when there had been a profuse discharge. Two days before admission she had had annoying eye trouble and was inclined to be stuporous.



Examination on admission: Patient semiconscious; temperature  $104^{\circ}$ ; pronounced clinical signs of mastoiditis, including tenderness, swelling, broken down posterior superior wall, and profuse discharge.

The examination of the urine showed acetone and a trace of albumin. The following day a simple mastoidectomy was done, the mastoid being distinctly pneumatic, great bone destruction, pus everywhere, a perisinus abscess (the sinus seemed to be functioning), necrosis of the tegmen antri and extradural abscess. The dura appeared relatively normal.

Culture from the pus of mastoid showed the streptococcus. The paralysis of the sixth nerve, which was present on admission, and probably two days before, had cleared within two weeks after the operation.

Four days after the operation erysipelas developed. As a further complication, eight or ten days later, cystitis became active. The mastoid wound healed in from seven to eight weeks.

Case 20 (Courtesy of Dr. R. E. Robinson).—The patient, a child of six years, had a history of a severe head cold, with earaches, the last of December, 1921. When seen early in March, 1922, there was a history of nausea and vomiting. The temperature was normal, the ear drum was red and bulging in the upper posterior quadrant, the hearing about normal, and a paralysis of the sixth nerve in evidence.

An X-ray picture of the mastoid was taken. This showed breaking down of the cell walls. There was tenderness on pressure over the mastoid, but no other clinical signs of mastoiditis. The next day a simple mastoid operation was done. The mastoid proved to be very large celled, pneumatic in type, with extension in every direction, and these were filled with granulations and very little free pus.

Following the operation there was uninterrupted convalescence, the paralysis disappearing in from two to three weeks.

Case 21 (Courtesy of Dr. Robert J. Hunter).—J. W., male, age 12, admitted to the hospital May 31, 1924.

Chief complaint: Profuse discharge from the left ear, with diplopia and severe head pains.

Family history: Father and mother living and well.

Past medical history: He has had the usual diseases of childhood.

History of present illness: One month ago he had an accumulation of cerumen removed from the left ear. Two weeks ago he had severe bilateral earache; shortly after the left ear began to discharge pus. For the past week he has had severe frontal headache on the left side. Anorexia has been present. Diplopia developed two days ago.

Examination: A general examination of the heart, lungs and abdomen, negative.

Ears: Right ear, negative; left ear, free purulent discharge and well marked clinical symptoms of acute mastoiditis.

Nervous system: A positive Kernig; reflexes depressed; rigidity of neck and paresis of left external rectus.

Eyes: O. D., media clear; disc margins well defined; macula and periphery negative; no optic neuritis, papilledema or gross fundus lesion. O. S., same as right.

June 1, 1924. A thorough simple mastoidectomy was done today. The mastoid cells were filled with granulation tissue and free pus. The bone over the sinus was in good condition, as well as the tegmen of the mastoid and antrum, so that there being no intracranial leads the sinus and dura were not uncovered.

June 2, 1924. Dr. J. W. McConnell saw the patient in consultation and confirmed the diagnosis of meningitis.

June 4, 1924. Bárány reactions of the right ear show horizontal and rotary nystagmus; past pointing not tried; fistula symptom the left ear, negative.

June 6, 1924. Marked convulsive seizures followed by death.

The patient ran a steeped temperature during the week he was under observation in the hospital.

Laboratory findings: Blood: White blood cells, 25,800. Urine: negative throughout. Culture of pus from the mastoid: *Bacillus mucosus capsulatus*, and a few *micrococcus catarrhalis*. Spinal fluid: The spinal fluid was obtained with difficulty in small amount. There were 915 cells per mm.; 72 per cent polys. Smears from the spinal fluid negative for bacteria. Cultures from the spinal fluid June 2nd and June 5th, negative for bacteria.

Final diagnosis and cause of death, acute meningoencephalitis. An autopsy could not be obtained.

Case 22 (Courtesy of Dr. Robert J. Hunter).—G. X., male, age 28. The patient had suffered for a number of weeks with a severe acute suppurative otitis media. No history was ascertained of any predisposing cause, such as head cold, influenza or sore throat. There had been spontaneous rupture of the tympanic membrane and a very free purulent discharge. There had been no attempt at treatment and the disease had pursued its own course until it came under observation of the writer. At this time there was a profuse discharge from the left ear, accompanied with all the clinical signs of an extensive mastoiditis. There was a complete paralysis of the left external rectus muscle. The signs of a suppurative meningitis were also in evidence. On account of the patient's extreme condition it seemed wiser not to operate, and death rapidly followed.

An autopsy could not be obtained.

Case 23 (Courtesy of Dr. William Arrell).—Mrs. H. T. came to me April 19, 1924. She has always been strong and healthy. She became ill March 1, 1924, when she had a chill. She had a slight cold before the chill. She had pain in the right ear about the third day after the chill, and on the fourth day the ear began to discharge. Pain continued in the ear and over the right side of head, face and brow, and continued to get worse. She could not eat or sleep much, felt ill and was unable to sit up. Discharge from ear was very profuse. She had some rise of temperature every day. She was attended by family doctor, who advised syringing ear.

When the patient came to my office for examination she happened to try to look to the right, and I noticed she could not rotate the right eye out. On further examination I saw there was complete paralysis of the right external rectus. There was tenderness and swelling over the mastoid.

The patient was prepared for operation as soon as possible. The mastoid was deeply involved.

The patient made a good recovery. Paralysis of the right external rectus cleared up in about two weeks and there was no diplopia. The ear continued to discharge for four weeks after operation.

Peculiarity: Pus came from canal very freely as well as from the mastoid wound.

The ear now gives no trouble and hearing is fairly good.

Case 24 (Courtesy of Dr. S. B. Marlow).—E. S., age seven years, was admitted to St. Joseph's Hospital March 10, 1922. Service of Dr. H. G. Kline.

Diagnosis: Suppurative otitis media right. Acute mastoiditis right. Paralysis external rectus, right and left.

History: For three or four weeks prior to admission the right ear had been discharging. The patient having had no treatment, the drum apparently had ruptured spontaneously. She now complained of pain in her head and in and about the right mastoid. The right eye had suddenly turned in, which condition alarmed the mother and led her for the first time to call a physician, who immediately referred her to the hospital.

Examination: On admission the patient was seen by Dr. Ayer, whose examination was essentially negative except for the paralysis of the right external rectus and the discharging right ear. Lumbar puncture done at this time resulted in a bloody fluid, the last tube of which was clear, 12 c. c. being removed under slightly increased pressure. No leucocytosis was present in the clear fluid. On admission: Rectal temperature, 101; pulse, 120; respiration, 24.

Ophthalmic examination: S. B. M. Except for the paralysis right external rectus essentially negative. The margins of both O. D. were blurred but not pathologic.

X-ray of mastoid: Dr. D. S. Childs. Right mastoid shows some increase in markings and density, but I do not feel that there is enough evidence on the film at this time to warrant operative measures unless there is striking clinical evidence. The cells extend into the zygomatic process and the lateral sinus is in normal position. Left side apparently within normal limits. Urinalysis, negative. W. B. C., 13,000; polys., 88 per cent; lymph., 12 per cent. Culture of discharge, negative.

Course: In view of the good clinical condition of the patient boric irrigations of the right ear were ordered and the patient kept under observation. Under this treatment the discharge subsided, and the temperature became essentially normal; the patient comfortable, sleeping well. Three days after admission she began to complain of pain in her right ear with headache

and became very restless. This condition so rapidly grew worse that operation was decided upon and performed at 2:45 a. m., March 14. Under general anesthesia the right mastoid was opened. In view of the right external rectus involvement the excavation was carried as deep as possible into the petrous position of the temporal bone. Following the operation the patient went through a rather stormy period, the temperature rising to 105.8° at one time. She was very dull, incontinent and had two convulsive attacks for which chloroform was administered. W. B. C., 30,000; polys., 88 per cent.

March 15th. Examination by Dr. Ayer was essentially negative except for questionable slight rigidity of the neck. Lumbar puncture was suggested but not done because of the condition of the patient, the hyperpyrexia being considered the explanation of the symptoms. The temperature gradually subsided, the clinical condition improving. However, at 9:30 p. m., March 15th (i. e., about 42 hours after the operation), the left eye was noted to be convergent.

March 16th. Examination by Dr. Ayer. No evidence of meningitis.

Ophthalmic examination: Right external rectus completely paralyzed as before. In addition the left was deviated in and could not be rotated beyond the midline to the left. No ophthalmoscopic changes. The patient's clinical condition otherwise continued to improve. Cultures taken from the mastoid at operation were reported staphylococcus aureus.

March 20th. Improvement continues. The outward rotation of the left eye was noted as nearly normal. That of the right definitely more than previously.

The convalescence was subsequently uneventful, the activity of the left external rectus became completely restored. She was discharged from the hospital 17 days after the operation. The right eye, however, was still slightly convergent. About two months after she left the hospital, when she was last seen, the right external rectus was still slightly underacting. Complete recovery of this muscle took place later and the child has since, as far as known, remained well.

Case 25 (Courtesy of Dr. S. B. Marlow).—Mr. P. P. was admitted to St. Joseph's Hospital July 2, 1921. Service of Dr. H. G. Kline.

Diagnosis: Suppurative otitis media left. Acute mastoiditis left. Paralysis external rectus left.

History: The patient was admitted to the hospital with the history of a discharge from his left ear for two weeks. For this he had been cared for by his physician, who finally sent him to the hospital because of the recent occurrence of severe pain in this ear and very definite mastoid tenderness.

Examination: Essentially negative except for discharging left ear, the drum of which had been ruptured and the definite mastoid tenderness. X-ray of mastoid: Dr. D. S. Childs. Right mastoid: well formed cells that extend well into zygomatic process. Emissary vein high. Lateral sinus in normal position. Left side shows lack of definition of cells and haze over left mastoid region. Diagnosis, left mastoiditis with some destruction of cells. On admission, temperature, 98; pulse, 90; respiration, 22.

Operation: July 2nd, 6 p. m. Under general anesthesia left mastoid exenterated.

Course: Convalescence uneventful, condition gradually improving, considerable discharge coming from left ear, however; on the ninth day the patient first complained of a diplopia for distance. Ophthalmic examination, seven days later by Dr. D. E. Gillette: Paresis left external rectus. Fundi normal. The patient's general condition continued to improve. The discharge from his ear persisted for some time, but finally stopped rather abruptly. The patient was discharged from the hospital on the twenty-eighth day after admission. The muscle paresis cleared after a time and the patient returned to work. He subsequently has remained well.

Case 26 (Courtesy of Dr. H. R. Randall).—Carl T. B., 8 years of age.

Previous history: Lobar pneumonia four years ago with a complication of bilateral otitis media. Recovery uneventful.

Present history: October 27, 1924, the patient was taken with an acute rhinitis and earache, which continued for two days, when rupture of the left drum occurred spontaneously. On October 30th, the patient was referred to me by Dr. Wilde. The drum was bulging and a profuse purulent discharge was in the external canal. Under chloroform anesthesia the drum was incised and irrigation started. Temperature ranged around

100 degrees and slight pain persisted in spite of free drainage. On November 4th, the patient was admitted to the hospital with temperature  $101.2^{\circ}$ , tenderness over mastoid, some edema and a drooping of the posterosuperior canal wall. X-ray showed a normal mastoid on the right with well defined cells. Left showed haziness of the whole, increasing to density over the antrum; all cells poorly defined, especially anterior to sinus wall. Operation was advised but parents preferred to wait a few days. Signs and symptoms continued with some increase. Operation on November 8th, simple mastoidectomy. All cells filled with pus with breaking down of the trabeculae in the region of the antrum. Culture was a gram positive diplococcus. Forty-eight hours after operation temperature became normal and remained as such for ten days, when patient was allowed to go home. Wound granulated well, packing of iodoform gauze was continued. External canal dry on third day. Dura was not exposed nor lateral sinus.

Four days after return home he complained of left temporo-parietal pain, lack of appetite and general malaise without temperature change. Occasional attacks of vomiting immediately after eating. Ten days later, on November 28th, complained also of pain back of left eye and difficulty in seeing. Referred him to Dr. J. D. Bowen, who reported: Patient tilted head to right to avoid diplopia.

O. D.: Ocular movement normal. Pupils 6 mm. react to light and accommodation; conjunctiva, negative. Ophthalmoscopic: Papilla round, margin sharply defined. Color normal; physiologic cup present. Retina and vessels normal.

O. S.: Limitation of outward movement at midline. Pupil reacts to light and accommodation. Conjunctiva normal. Ophthalmoscopic: Papilla normal, color, margin distinct and sharply defined; physiologic cup present; retina normal, also vessels.

Diagnosis: Strabismus paralytic left external rectus. Patient confined to bed. Internist saw patient and reported negative findings; recommended spinal puncture, but parents refused consent at that time. Pronounced lack of appetite and pain of parietal area continued. Temperature continued normal with occasional subnormal remissions. Blood count showed on December 4th: Leucocytes, 7,400; erythrocytes, 4,100,000; Hbg.,

75 per cent; color index, .91, 66 per cent; polynuclears, 27 per cent; lymphocytes, 4 per cent; large mono, 2 eosinophiles.

Signs and symptoms continued. Postauricular wound healed entirely on December 12th, when lumbar puncture was done. Fluid pressure of 7 mm. Laboratory reported cell count 3. Sugar slight trace. Globulin positive. Differential polys, 35 per cent; small lymphocytes, 57 per cent; large, 6 per cent; transitional, 2 per cent; smear and culture negative.

Pain continued in parietal area and back of eye. He was encouraged to eat and move about and gradually began to feel better. Mild sedative only was used as medication with tincture of nux as a tonic. January 2, 1925, some motion was noted of external rectus of left eye.

January 13th, Dr. Bowen again reported: Outward movement of the left eye is greater than seen on January 2nd. At the previous examination the outer limit of movement was to within 5 mm. of the outer canthus. At present the outward movement of the limbus is within 1 mm. of the external canthus.

Pain has entirely stopped, appetite has returned and he is now back in school. There was no evidence of labyrinthine disturbance at any time. The only reasonable explanation apparent was a localized serous meningitis or a toxic neuritis, if this is a case with the Gradenigo syndrome. If there was an abscess it was very small and did not cause a change in the spinal fluid or pressure symptoms.

Case 27 (Courtesy of Dr. Andrew Hunter).—Anthony C., age 35, developed a left middle ear infection about February 1st. Before I saw him he had developed an abscess back of the left ear, which had been opened by the family physician. He had a great deal of pain in the head, in spite of a free discharge from the opening back of the ear, which probing showed communicated with the mastoid. At this time he also complained of pain over the left maxillary sinus and evidently had an infection in this locality also, for a dentist had removed some teeth which penetrated the antrum; this was curetted.

I did a simple mastoid operation on him March 5th, during which the cells of the tip were found necrotic (a great deal of necrosis), but there was no necrosis in the neighborhood of the mastoid antrum. The mastoid wound was treated by



daily dressing and the patient rapidly improved, although he complained of some headache. He came to my office for dressing of the mastoid wound on April 5th, and at that time a complete paralysis of the left external rectus muscle was present. His vision was 6/6 in either eye, there was no swelling of the optic discs, there was no headache and there was no diplopia. The patient had had some trouble with his left eye for two weeks previous to this visit. At this time the patient was in fairly good health, with good appetite and apparently recovering, in the usual way, from a mastoid abscess.

April 7th, I was called in the morning to see the patient. On the previous day he had been well in the morning, ate breakfast and lunch, then went down street to his place of business. He fell sick in the afternoon, returned home, vomited twice, threw himself upon the bed, and did not get up afterwards. When I saw him on the morning of April 7th he had pain in the head and back and rigidity of the muscles of the neck. Meningococci were found in the spinal fluid. He died at 8:50 p. m.

Analysis of 172 cases of Abducens Paralysis taken from 126 authors, with reference to: Occurrence in Acute or Chronic Otitis, Age, Sex, Side, History of Predisposing Cause, Pain, Opening of Tympanic Membrane, Time of Appearance of Paralysis, Time of Disappearance of Paralysis Relative to its Appearance, Recovery, Improvement, or no Improvement of Paralysis, Character of Operation, Operative Findings, Optic Nerve Changes, Spinal Fluid Changes, Pathogenic Organisms Present, Roentgen Ray Findings, and Causes of Paralysis given by various Authors.

# OCCURRENCE IN ACUTE OR CHRONIC OTITIS.

OTITIS.		Not Given						
Acute	Chronic							
125	29	18						
1-10	10-20	20-30	30-40	Age 40-50	50-60	60-80	Not Given	
28	37	26	16	15	4	1 (72)	45	
Male	Female	Not Given		Sex				
76	55	41						
Right	Left	Bilateral	Opposite Side	Side Not Given				
62	56	6	14	46				

# HISTORY OF PREDISPOSING CAUSE.

Influenza 13 29	Head Cold 18 Not Given 88	Sore Throat 7	Measles 2	Scarlet Fever 5	Pneumonia 1	Puerperal Sepsis 1	Acute Illness in General 2
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## PAIN.

Present 129	Not Present 2	Severe 121	Moderate or Slight 8	Not Given 41			
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# OPENING OF TYMPANIC MEMBRANE.

Spontaneous Perforation 39	Incision 24	Old Perforations and partly Destroyed 20	Not Given 50				
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## TIME OF APPEARANCE OF PARALYSIS.

AS TO DAY OF OTITIS.		AS TO OPERATION.		AS TO OPERATION.		AS TO OPERATION.	
1-10 17	10-20 37	20-60 29	Two Months or More 31	Not Stated, Pres- ent at First Visit 1	During Operation 43	After Operation, Time not Stated 8	Not Given 8

## TIME OF DISAPPEARANCE OF PARALYSIS RELATIVE TO ITS APPEARANCE.

AS TO DAY OF OTITIS.		AS TO OPERATION.		AS TO OPERATION.		AS TO OPERATION.	
1-10 9	10-20 15	20-60 30	Two Months or More 22	Not Stated, Pres- ent at First Visit 1	During Operation 16	After Operation, Time not Stated 16	Not Given 16

# RECOVERY, IMPROVEMENT, OR NO IMPROVEMENT OF PARALYSIS.

Complete	Improved	Unimproved	Deaths	Not Given	
109	10	3	31	20	

## CHARACTER OF OPERATION.

None 10 Incision of Tym- panic Membrane Without Other Operation 7	MASTOIDECTOMY.				Lateral and other Sinus 19	Ligation and Re- section of Int. Jug. Vein 16	Exploration of Brain 16
	Simple 111 Incision of Exter- nal Ostitis	Radical 44 Nasal Sinus With- out Ear Operation	Secondary Operation 30 Exenteration of the Labyrinth	Exposed Dura 37 Not Given			
	1	1	1	9			

## OPERATIVE FINDINGS.

Cellular 64 BRAIN ABS- CESSSES. Extracranial 11	STRUCTURE OF MASTOID.		Not Given 73 Retropharyngeal Abscess 2	Pus with Slight Granulation 55 Purulent Labyrinthitis 1	Granulation with Little Pus 48	Bone Destruction 51	Sinus Thrombosis 27
	Diploetic or Sclerotic 25 Intracerebral 10	Not Operated 16 Nasal Sinus Pus and Granulation 6					

## OPTIC NERVE CHANGES CLASSED AS OPTIC NEURITIS OR PAPILLEDEMA AND CHOKED DISC.

Optic Neuritis 13	Papilledema or Choked Disc 24	Not Present 67	Not Given 68	
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# SPINAL FLUID AS TO PRESSURE AND CELL COUNT.

Pressure Increased 34	Not Increased 19	Cell Count Increased 14	Not Increased 16	Not Given 119		
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## PATHOGENIC ORGANISMS PRESENT.

STREPTOCOCCI						
Not Typed	Streptococcus Muc. Cap.	Hemolyt.	Pneumococcus Not Typed	Staphylococcus Albus	Mixed Streptococcus, Staphylococcus and Pneumococcus	Bacillus Klebs. Loefler
13	11	4	5	2	7	1
Bacillus Colon	No Growth on Culture					Bacillus Influenza
1	2					2

## ROENTGEN RAY FINDINGS.

Reported variously, character of mastoid, cloudiness or haziness of cells, bone necrosis, breaking down of intercellular walls, repair of bone, position of sinus.

## CAUSES OF PARALYSIS GIVEN BY VARIOUS AUTHORS.

Circumscribed Pachymeningitis at Apex of Petrosal Sinus	Serous or Prolfective Meningitis with Pressure on the Sixth Nerve	Inflammatory Edema at the Apex of Pyramid	Extradural Abscess	Toxic Neuritis	Edema of Brain with Increased Intracranial Pressure on Sixth Nerve	Extension of Inflammation from Lateral Sinus or Bulb to Region of Sixth Nerve	Encephalitis
19	9	4	2	16	7	4	1
Thrombosis of Superior Petrosal Sinus	Not Given	Abscess at Apex of Petrosal Sinus	Hemorrhage into Brain Substance				
1	105	4	1				

AUTHORS OF 26 UNPUBLISHED CASES, TOGETHER WITH  
ONE BY THE AUTHOR IN 1909.

- Case 1. Sears, Wm. H., Huntingdon, Pa.  
 2. Sears, Wm. H., Huntingdon, Pa.  
 3. Sears, Wm. H., Huntingdon, Pa.  
 4. Ersner, M. S., Philadelphia, Pa.  
 5. Ersner, M. S., Philadelphia, Pa.  
 6. Goddard, H. M., Philadelphia, Pa.  
 7. Goddard, H. M., Philadelphia, Pa.  
 8. Colver, B. N., Battle Creek, Mich.  
 9. Clark, Ivor, Columbus, Ohio.  
 10. Clark, Ivor, Columbus, Ohio.  
 11. Weinberger, N. S., Sayre, Pa.  
 12. Kreiger, W. A., Poughkeepsie, N. Y.  
 13. Cobb, Edwin, Marshalltown, Iowa.  
 14. Sullivan, Jno. J., Jr., Scranton, Pa.  
 15. Beck, Jos. C., Chicago, Ill.  
 16. Weinberger, N. S., Sayre, Pa.  
 17. Shambaugh, Geo. E., Chicago, Ill.  
 18. Fischer, N. A., Pittsburgh, Pa.  
 19. Robinson, R. E., Waverly, Iowa.  
 20. Robinson, R. E., Waverly, Iowa.  
 21. Hunter, Robert J., Philadelphia, Pa.  
 22. Hunter, Robert J., Philadelphia, Pa.  
 23. Arrell, William, Hamilton, Ontario.  
 24. Marlowe, S. B., Syracuse, N. Y.  
 25. Marlowe, S. B., Syracuse, N. Y.  
 26. Randall, H. R., Binghamton, N. Y.  
 27. Hunter, Andrew, McKeesport, Pa.

BIBLIOGRAPHY AND REFERENCES ASSOCIATED WITH  
THE 172 CASES REPORTED AND TABULATED.

1. Gradenigo: Arch für Ohrenheilk., Vol. 74, p. 149. 1907.
2. Perkins, Chas. E.: Annals Otol., Rhinol. and Laryngol., 1910, p. 694.
3. Kollner: Deutsch. med. Wochen. 1908. No. 3; p. 112.
4. Cushing: Brain, Vol. XXXIII, p. 204.
5. Wheeler, Jno. M.: Transactions Sect. Oph. A. M. A. 1918.
6. Vail, H. H.: Laryngoscope. August, 1922. Vol. XXXII, No. 8, p. 569.
7. DuPuy, Homer: Southern, M. J. 17:213. March, 1924.
8. Collet, F. J: Arch. Internat. de Laryngol, etc. Paris—May, 1923.
9. Rutlin, Eric: The Laryngoscope, Nov., 1911, p. 1051.
10. Auerbach and Alexander: Mittheilungen a. d. Grenzgebiet der Med. u. Chir. 1913. Vol. 25, p. 431.
11. Eagleton: Brain Abscess (Macmillan Co.) 1922.
12. Sullivan: Case Report 14.
13. Wilkinson, Geo.: Journal Laryngology, Otology and Rinology. 1914. Vol. XXIX, p. 409.
14. Eklund, Thure: Arch. für Ohren, Nasen and Kehlkopfheilk. 1917—May.

15. Dench, E. B.: *Annals Otol., Rhin and Laryng.* Sept., 1916, p. 672.
16. Roy D.: *Annals. Otol., Rhin. and Laryng.* March, 1921, p. 244.
17. Quadri: *Revue Hebdomadaire de Lar., etc.* 1908. Vol. 28, part 2, p. 100.
18. Sluder: *Trans. Am. Laryng. Soc.* 1912.
19. Onodi: *Arch für Laryng.* 1912. Bd. 26, Heft 2.
20. Onodi: *Journal of Laryng., Rhin. and Otol.* XXIX, p. 304.
21. Fremel, F.: *Monat. für Ohrenh., etc.* Berlin. 56:230—April, 1922.
22. Loughran, Robert: *Annals of Otology, Rhin. and Laryng.* June, 1916; p. 497.
23. Smith, J. Morrisett: *Medical Record.* p. 18; Vol. 95; No. 1.
24. Page, J. R.: *Annals of Otology, Rhin. and Laryng.* June, 1916, p. 497.
25. De Lamothe: *Revue de Laryngol.* Paris, 1920, XLI, 198.
26. Snee, Harry Boyd: *Annals of Otology, Rhin and Laryng.* Sept., 1924.
27. Dunlap, A. M.: *Laryngoscope.* Oct., 1922. Vol. 32.
28. Knick, Arthur: *Zeitschrift für Hals, Nasen and Ohren.* 1922, p. 136.
29. Alt: *Monats für Ohrenheilk.* 40. Jahrg.
30. Fremel, F.: Vienna, Austria. Personal communication.
31. Shemely, W. G.: *Annals of Otology.* Sept., 1922.

The above are listed in the order in which reference is made; the following are arranged chronologically except from No. 109 to 138.

32. Smith, E. Terry: *Trans. Am. Otological Soc.* Vols. 9-10; 1905-07. p. 551.
33. Eagleton, Wells: *Ibid.* p. 553.
34. Collinet: *Bull. d. Laryngol., Otologie and Rhinologie.* X 90. 1907.
35. Welner, Anna S.: *Archives Pediatrics.* Jan., 1907. p. 18.
36. Jones, Clarence P.: *Laryngoscope.* July, 1908.
37. Blaululet: *Report of Société Parisienne d'Otologie.* 1909.
38. Kenefick: *Annals of Otology, Rhin. and Laryng.* 1910-19—p. 710.
39. Lehmann, Richard: *Deut. med. Woch.* 1910. No. 29. p. 1368.
40. Leto, L.: *Boll. della Mal. dell' Orrechio della Gola, etc.* April, 1911. p. 75.
41. Farnarier: *Marseilles Méd.* No. 2. 1911.
42. Ruttin, Eric: *The Laryngoscope.* Nov., 1911. p. 1051.
43. Savareaud and de Lamothe. *Bull. d'Otologie, Rhino-Laryngol.* XV. 206. 1912.
44. Graham, H. B.: *Laryngoscope;* 1913, Dec. p. 1147.
45. Hays, Harold: *Annals of Otology.* June, 1913. p. 514.
46. Auerbach and Alexander: *Mittheilungen, a. d. Grenzgebiet der Med. u. Chir.*
47. Bartels: 1913. Vol. 25. p. 431. a.
48. Muecke, F. F.: *Jour. Laryng., R. and O.* 1914. p. 77.
49. McNab: *Journal of Laryngology.* Sept., 1915. p. 335.
50. Meyers, H. L.: *Annals of Otology, Rhin. and Laryng.* Vol. XXIV. p. 800. 1915.
51. Friedenwald and Downey: *Trans. of Am. Otol. Soc.* 1915. p. 503.

52. Guentzer, J. H.: *Annals of Otology, Rhin. and Laryng.* June 1916. p. 490.
53. Friesner; *Ibid.* p. 493.
54. Dench, E. Bradford: *Ibid.* Sept., 1916. p. 672.
55. Guentzer, J. H.: *Ibid.* June, 1916. p. 499.
56. Coates, George M.: *Ibid.* June, 1917. p. 409.
57. Guthrie, D.: *Edinburgh Med. Journal.* July, 1917.
58. Eagleton, Wells: *Brain Abcess* (Macmillan Co.), 1922, p. 38.
59. Dighton, Adair; *Annals of Otology, Rhin. and Laryng.* June, 1917. p. 554.
60. Wheeler, John M.: *Trans. Sect. Oph., A. M. A.* 1918.
61. Jansen and Kobrak: *Practical Otology for Physicians.* 1918. p. 248.
62. Jansen: *Ibid.* p. 249.
63. Smith, A. E.: *Minnesota Med.* 1919. No. 2. p. 424.
64. Oppenheimer, Seymour: *Laryngoscope.* March, 1919. p. 172.
65. Tanaka, H.: *Laryngoscope.* August, 1919. p. 491.
66. Friesner, Isidore: *Annals of Otology, Rhin. and Laryng.* March, 1919, p. 90.
67. Steckney, Otis: *Laryngoscope.* 1919. Vol. XXIX, No. 7.
68. White, E. H.: *Am. Jour. of Med. Sciences.* 1920. February. p. 227.
69. Maybaum, J. L.: *Laryngoscope.* 1920. March. p. 138.
70. Perkins, Charles E.: *Laryngoscope.* October, 1920. p. 666.
71. Hill, Frederick T.: *Annals of Otology, Rhin. and Laryng.* Dec., 1920. p. 850.
72. Page, John R.: *Ibid.* Sept., 1920. p. 745.
73. Zentmayer, William: *Am. Jour. Oph.* Oct., 1920. p. 766.
74. Kan, T. L.: *Nederl. Tijdsch. v. Geneesk.* 1920. p. 857.
75. Marin Amat: *Rv. Cubana de Oft.* 1920. V. 2. p. 820.
76. Eagleton, Wells: *Annals of Otology, Rhin. and Laryng.* June, 1920. p. 499.
77. DeLamothe: *Revue de Laryngol.* Paris, 1920. XLI, 198.
78. Urbantschitsch: *Wien. klin. Woch.* 1920. V. 33. p. 166.
79. Amberg, Emil: *Detroit Med. Jour.* May, 1920.
80. Ditman, George C.: *Minnesota Medicine.* III; p. 439. 1920.
81. Mollison, W. M.: *Brit. Jour. Children's Dis.* July-Sept., 1921. pp. 18-135.
82. Meleney, Henry E.: *Laryngoscope.* Oct., 1922. p. 763.
83. Norcross, E. P.: *Annals of Otology, Rhin. and Laryng.* June, 1922.
84. Maicanescu, M.: *Sptalul.* Vol. 42. p. 7. Bucharest, 1922.
85. Adams, C. J.: *Annals of Otology, Rhin. and Laryng.* Dec., 1922. 31:984.
86. Knick, Arthur: *Zeitschrift für Hals, Nasen and Ohren.* 1922. p. 136.
87. Grosman, Fritz: *Ibid.* p. 145.
88. Link: *Königsberg.* *Ibid.* p. 146.
89. Voss: *Frankfurt Ibid.* p. 147.
90. Schenke: *Flensburg.* *Ibid.* p. 147.
91. Werner: *Mannheim.* *Ibid.* p. 148.
92. Eagleton, Wells: *Brain Abcess* (Macmillan Co.), 1922.
93. Simpson, J. R.: *Atlantic Med. Jour.* May, 1923. p. 549.
94. Friedenwald and Breitstein: *Laryngoscope.* Nov., 1923. p. 820.



95. Lapouge: *Rev. de Laryngol.* Bordeaux. 44:763. Sept. 30, 1923.
96. Bowers, W. C.: *Laryngoscope.* June, 1923. p. 462.
97. Downey, Jesse W.: *Internat'l Survey Oph. and Oto.* Laryng. Oct., 1923.
98. Chamberlin, W. B.: *Atlantic Med. Jour.* June, 1924. p. 569.
99. Roberts, E. L.: *Laryngoscope.* April, 1924. p. 319.
100. Kelson, W. H.: *Eye, Ear, Nose and Throat Monthly.* July, 1924. p. 350.
101. Just, T. H.: *Ibid.*
102. Scott, Sydney: *Ibid.*
103. Tweedie, A. R.: *Ibid.*
104. Profaut, Henry J.: *Laryngoscope.* Feb., 1924. p. 140.
105. King, J. J.: *Laryngoscope.* July, 1924. p. 578.
106. Phillips, A. Kenneth: *Journal Lancet*, 44:165. March 15, 1924.
107. Neubauer, Adolf: *Jahrb. für Kinderhkl.* Berlin. 104:361. April, 1924.
108. Day, Ewing: *Pittsburgh, Pa. Atlantic Med. Jour.* June, 1924. p. 572.
109. Fridenberg, P.: *New York City. Arch. f. Ohren.* 1907, p. 45.
110. Tomassi: *Annali Ital. di Larynge Otol.* No. 6. 1908.
111. Royce: *Laryngoscope*, 1909, p. 629.
112. Broeckhart: *Revue de Larynge., etc., Paris.* 1915. XXXVI, p. 209.
113. Jacques: *Revue de Larynge., etc., Paris.* 1915. XXXVI, p. 233.
114. Caldera: *Arch. Ital. di Otologia.* Napoli. 1918. XXXVIII. p. 321.
115. Aboulker, Henri: *Revue de Laryngol., etc., Paris.* Dec. 15, 1921. 23:649.
116. Nicolo, Filippi: *Arch. Ital. di Otologia.* Napoli. Oct., 1922. 33:302.
117. Chavanne: *Zentralblatt für Ohrenheilk.* 1920. Bd. 18, s. 16.
118. Markiefka: *Dissertations of Rostock.* 1909.
119. Yzeriman: *Zentralblatt für Ohrenheilk.* 1917. Bd. 14, p. 240.
120. Gavello: *Monatschr. für Ohren.* Bd. 45, s. 814.
121. Fischer: *Monatschr. für Ohren.* Bd. 52, s. 532. 1919.
122. Dinalt: *Monatschr. für Ohren.* Bd. 52, s. 533. 1919.
123. Bondy: *Monatschr. für Ohren.* Bd. 52, s. 533. 1919.
124. Urbantschitsch, E.: *Monatschr. für Ohren.* 1919. Bd. 52, s. 533.
125. Frey, Hugo: *Monatschr. für Ohren.* 1919. Bd. 52, p. 534.
126. Prade: *Monatschr. für Ohren.* 1912. Bd. 46, p. 1423.
127. Kerandren: *Arch. für Ohren.* Bd. 88, s. 114.
128. Anthony, D. H.: *Amer. Jour. Oph.* 1923. Sept. p. 773.
129. Uffenorde: *Arch. für Ohren.* 1920. Bd. 105, s. 87.
130. Neuman: *Monatschr. für Ohren.* 1911. Bd. 45-1, s. 239.
131. Alexander, G.: *Arch. für Ohren.* 1912. Bd. 89, s. 256.
132. Balla, Alberto: *Zentralblatt für Ohrenheilk.* 1919. Bd. 17, s. 246.
133. Malan, A.: *Zentralblatt für Ohrenheilk.* 1911. Bd. 9, s. 265.
134. Nagaoka, Y.: *Zentralblatt für Ohrenheilk.* 1910. Bd. 9, s. 555.
135. Sterling: *Monatschr. für Ohrenheilk.* 1910. Bd. 44, s. 1045.
136. Leegeard: *Zentralblatt für Ohrenheilk.* 1919. Bd. 17, s. 98.
137. King, J. J.: *Transactions Am. Acad. Oph. and Otolaryng.* 1924. p. 281.
138. Ulrich, K.: *Zeit. f. Hals, Nasen u. Ohren.* 9:403. Jan. 20, 1925.

In addition to the 172 cases presented in this report the following list comprises practically all of the references which have appeared in the literature, including all of the authors given by Gradenigo and Perkins, as well as those given by other writers, and all of the cases the writer has found in the literature not mentioned in any bibliography:

1. Alt: Monatschr. für Ohrenheilk. 88, 1906.
2. Alt: Wien. med. Woch., Nr., 37, 1917.
3. Baldenweck: Thèse de Paris, 1908.
4. Balla: Zentralblatt für Ohrenheilk. 1920. Bd. 17; s. 240.
5. Barr: British Med. Jour., 1908. Part 2, p. 888.
6. Baratoux: Archivio Intern. de Laryng, etc. 1907.
7. Bartels: Monatschr. für Psychiat., 1910. Bd. 28; s. 85.
8. Baurowicz: Annales d. mal. l'or., 15. 1906.
9. Benoit: Thèse de Lyon.
10. Bezold: Lehrb. d. Ohr. 8. 1906.
11. Bielschowsky: Ergebn. d. Chir. u. Orthrop., 1916.
12. Billotti: L'Ospedale Maggiore di Milano, Feb., 1907.
13. Blanc: Annales de Mal. de l'Or., etc., 35, p. 192.
14. Bonnier: Presse Méd., 16. Dec., 1903.
15. Brieger: Enzyklopadie f. Ohr. s. 248. 1900.
16. Braunstein: Arch für Ohren., Vol. 55, p. 222.
17. Bruhl: Lehrb. u. Atlas der Ohrenheilk. 1913.
18. Burkner: Arch. für Ohren., 19, 1883.
19. Ceccaroni: Archivio Ital. di Otologia, etc. 18. 1907.
20. Cheval: Soc. Franc. d'Otologie. May, 1904.
21. Citelli: Archivio Ital. di Otologia, etc. 15, 404. 1904.
22. Citelli: Archivio Ital. di Otologia, etc. 18. 1907.
23. Cozzolino: Giorn. Intern. de Sci. Med. 1907.
24. D'Ajutolo: Arch. de Otol., Vol. 14, p. 210.
25. Denker-Brunings: Lehrb. d. Krankheiten d. Ohren, u. d. Luftwege. 1915.
26. Dorello: Atti della Clinica ot-laring di Roma, 1905.
27. Dorello: Zbl. f. Ohrenheilk. Bd. 8, s. 45. 1910.
28. Dorello: Monatschr. für Ohrenheilk. Bd. 46, s. 332. 1912.
29. Dundas Grant.
30. Forselles: Empyem des Warzen., etc., Liepsic. 1906; p. 63.
31. Gavello: Zbl. f. Ohrenheilk. Bd. 7, s. 207. 1909.
32. Gavello: Zchr. f. Ohrenheilk. Bd. 61, s. 81. 1910.
33. Gavello: Monatschr. f. Ohrenheilk. Bd. 45, s. 814. 1911.
34. Geronzi: Archivio Italiano di Otologia, etc. 1899. VIII, S. 1.
35. Geronzi: Archivio Italiano di Otologia, etc. 1905. XVI, 163.
36. Gervais: Thèse de Paris, 1879.
37. Gignoux: Rev. Hebdom. de Laryngol. Feb. 7, 1914. p. 161.
38. Goettermann: Deutsch. med. Wochenschr. Vol. 33. p. 552.
39. Goppert: Enzyklop. d. klin. Medizin. 1914. s. 107.
40. Goris: Annales de Mal. de l'Or. XIX. 1903.
41. Gradenigo: Arch. f. Ohrenheilk. 1912. Bd. 88, s. 294.
42. Gradenigo: Arch. f. Ohrenheilk. 1904. LXII, 255.
43. Gradenigo: Reale Accademia di Medicina in Torino. 29; Jan., 1904.
44. Gradenigo: Riunione della Società Ital. di Laring. e Otologia, Milano. 1906.

45. Gradenigo: Riunione della Societa Ital. di Laring. e Otologia, 1905.
46. Gradenigo: Naturforschervers. in Meran. Sept., 1905.
47. Grivot: Annales de Mal. de l'Oreille. 1908, p. 271.
48. Gruber: Tr. Soc. Austr. d'Otol. Nov. 24, 1895.
49. Guttich: Zeitschr. f. Laryngol. 1920. Bd. 9, s. 403.
50. Habermann: III Vers. d. deutschen otol. Gesellschaft. Jena, S. 94, 1898.
51. Hastings: Archives of Otology. 1906.
52. Hedon: Arch. Internat. de Lar. de Paris. Vol. 25, p. 412.
53. Hilgermann: Z. f. O., XI, 273. 1902.
54. Jack: Laryngoscope, 521. 1905.
55. Jacques: Annales de Mal. de l'Oreille, 15. 1906.
56. Jurgensmeyer: Vers. d. deutschen otol. Gesellschaft, Jena. S. 94, 1898.
57. Katz: Vers. d. deutschen otol. Gesellschaft, Jena. S. 94. 1898.
58. Keller: Monatschr. für Ohrenheilk. XXI. 1888.
59. Knapp: Arch. Opth. Vol. 38, p. 552.
60. Kollner: Deutsch. med. Wohnschr., V. 7, p. 112.
61. Korner: Lehrb. d. Ohren. Nascn. u. Kehkopfkrankeheiten. 1918.
62. Korner: Die Otischen Erkrankungen di Otol. XIV. 210, 1903.
63. Kourteff: Thèse de Paris. 1907.
64. Lange: Passow-Schafers Beitr. 1909. Bd. 2, s. 162.
65. Lannois and Ferrand: Soc. Franc. d'Otologie. May, 1904.
66. Lannois and Perretoses.
67. Lapersonne: Zit. n. Sterling. Zbl. f. Ohrenheilk. 1914. Bd. 12, s. 99.
68. Lasagna: Zschr. f. Ohrenheilk. 1913. Bd. 11, s. 87.
69. Lecgaard: Zbl. f. Ohrenheilk. 1919. Bd. 17, s. 98.
70. Lehmann: B. kl. W. 1914, s. 295.
71. Leimer: Z. f. O., 242. 1904.
72. Lodge: Jour. of Laryng. London. Vol. 16, p. 536.
73. Lombard: Annales de Mal. de. l'Oreille, etc. 1906. Vol. 32, p. 321.
74. Lombard: Monatschr. für Ohrenheilk. 1910. Bd. 44, s. 1037.
75. Lubet Baron: Arch. Internat. de Lar., etc. de Paris. Vol. 18. p. 31.
76. Luc: Ref. in Gradenigo. 42. 1905.
77. Malan: Zbl. f. Ohrenheilk. 1911. Bd. 9, s. 265.
78. Mann: Arch für Ohrenheilk. Vol. 45, p. 121.
79. Martin: Rev. Barcelona de Enfr. di Oida. 1911. 6/30.
80. Mauthner: Zbl. f. Ohrenheilk. 1910. Bd. 8, s. 566.
81. Mayo-Collier: Journal of Laryngology, XVI, 536. 1901.
82. Meyer: A. f. O., XXXVIII, 258. 1895.
83. Modestini: Archivo Ital. di Otologia, etc., XVIII. 1907.
84. Molist: Contribucion a l'estudio clinicio, Barcelona, Badia Verl. 1905.
85. Mongardi: Archivo Ital. di Otologia, XV, 404. 1904.
86. Muck: Zeitsch. für Ohrenheilk. Vol. 37, p. 191.
87. Myers.
88. Nagaoka: Zbl. f. Ohrenheilk. 1911. Bd. 9, s. 555.
89. Nager: Zeitsch. f. Ohrenheilk. Bd. 53, s. 220.
90. Neumann: Zbl. f. Ohrenheilk. 1905. Bd. 3, s. 52.
91. Noltenius: Revue hebdom. de Laryng. 2; Dec., 1905.
92. Ostmann: Arch. Opth. (Graefe's) 1879. Vol. 43, p. 1.
93. Custom: Brit. Med. Jour. 1891. Part 1, p. 208.
94. Perretiese: Archives Intern. de Laryngol. XXI, 124. 1906.

95. Peyser.
96. Peyser, A.: B. kl. Wohnschr. 1908. No. 28.
97. Pick: Naturforschervers. in Meran. Sept., 1905.
98. Pischel: Z. f. Ohren. XL, 273. 1902.
99. Poli: Zbl. f. Ohren. 1907. Bd. 5, s. 108.
100. Poppi: Archivio Ital. di Otologia, etc., XVIII. 1907.
101. Prautois: Revue Méd. de l'Est, Vol. 24, p. 396.
102. Preysing: Z. f. Ohren, XXIII, s. 8. 1898.
103. Quadri: Revue Hebdom. de Lar., etc., 1908. V. 28, part 2, p. 100.
104. Rakowicz: Kl. Mbl. f. Augenheilk. 1895. Bd. 23, s. 163.
105. Ricci: Mongardi, Archivio Ital. di Otologia, XV; 404.
106. Rimini: Arch. Internat. de Lar., etc., Vol. 21, p. 125.
107. Schwarzkopf: Sammelreferat, Zentralblatt f. Ohren. Bd. Heft. 5. 1907.
108. Sauvinau: Annal. d'Ocul. 1907. p. 321.
109. Siebenmann: Siebenmann, Anatomie d. Sinnesorgane von Schwalbe. 1906.
110. Shulze: Arch. f. Ohren. 1903. S 1 U, S. 36, F. 7.
111. Spira: A. f. Ohren. XLI, 123. 1896.
112. Sterling: Monatschr. f. Ohrenheilk. 1910. Bd. 44, s. 1045.
113. Sterling: Monatschr. f. Psychiatrie. 1910. Bd. 34, s. 568.
114. Stottart: Journal of Laryngol. 1906.
115. Strazza: Archivio Ital. di Otologia, XVIII. 1907.
116. Streit: Arch. f. Ohren. Vol. 59, p. 169.
117. Sturm: Z. f. Ohren. XL, 273. 1902.
118. Styx: Zeitsch. für Ohren. 1898. Vol. 33, p. 8.
119. Terson: Annales de Mal. de l'Oreille, 15. 1906.
120. Tommassi: Bollettino della Società medica di Lucca. N. 3. 1905.
121. Tommassi: Archivio Ital. di Otologia, XVIII.
122. Torok: Annales de Mal. de l'Oreille, XIX, 64. 1903.
123. Torrini: Balle della Malorrechio. May, 1915.
124. Torrini: Arch. di Ital. de Otolog. January, 1919.
125. Triflelli: Archivio Ital. di Otologia. XV, 404. 1904.
126. Uchermann: Zbl. f. Ohren. 1918. Bd. 15, s. 63. Ber. Otol-Laryng. Verein Christiana.
127. Urbantschitsch: Lehrb. d. Ohr. s. 452.
128. Vizmanos: Sigle Med. 6/5, 1912. p. 371.
129. Woods: Journal of Laryngology XVI, 536. 1901.
130. Bourhut.

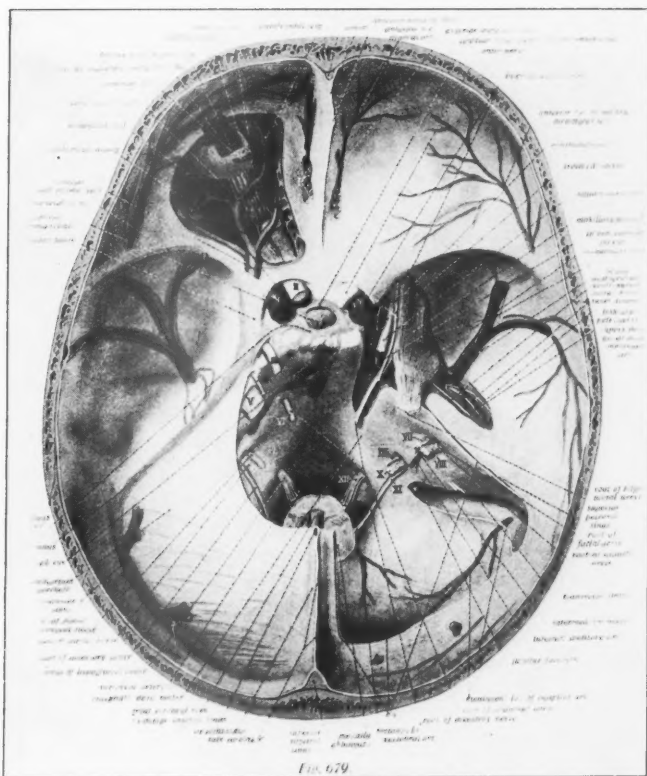


PLATE I.

From Sobotta McMurrich.

Showing on the right the relations of the nerves and carotid canal with its plexus in the cavernous sinus. The relation of the nerves from above downward and from before backward are the third, fourth, ophthalmic division of the fifth, and the sixth. The Gasserian ganglion is seen as it lies in the impressio trigemini of the petrous bone. On the left the sixth nerve pierces the dura below the inferior petrosal sinus, with which it lies in Dorello's canal. The superior wall of the cavernous sinus is well shown, as is also the internal end of the superior petrosal sinus, where it enters the cavernous sinus and lies over the Gasserian ganglion.

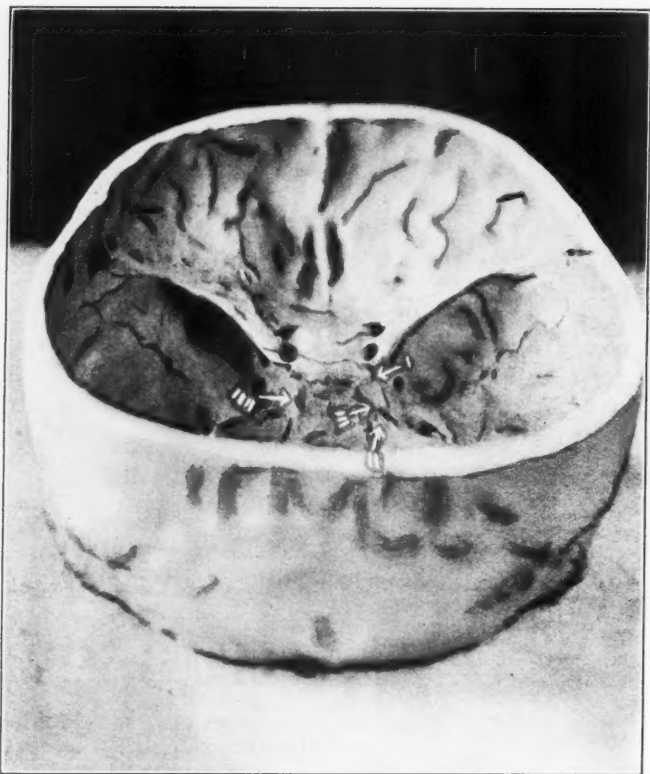


PLATE II.

Photograph of Skull.

Arrow I—Posterior clinoid process; Arrow II—the groove for the sixth nerve and just beyond the sphenoidal spine of the petrous; Arrow III—the point lies on the accessory posterior clinoid process (Dorello's canal placed below and between Arrows II and III); Arrow IIII points to the accessory posterior clinoid process on the left side.

Note the small size of Dorello's canal (on both sides); and also the smaller canal on the right (very marked in the skull).

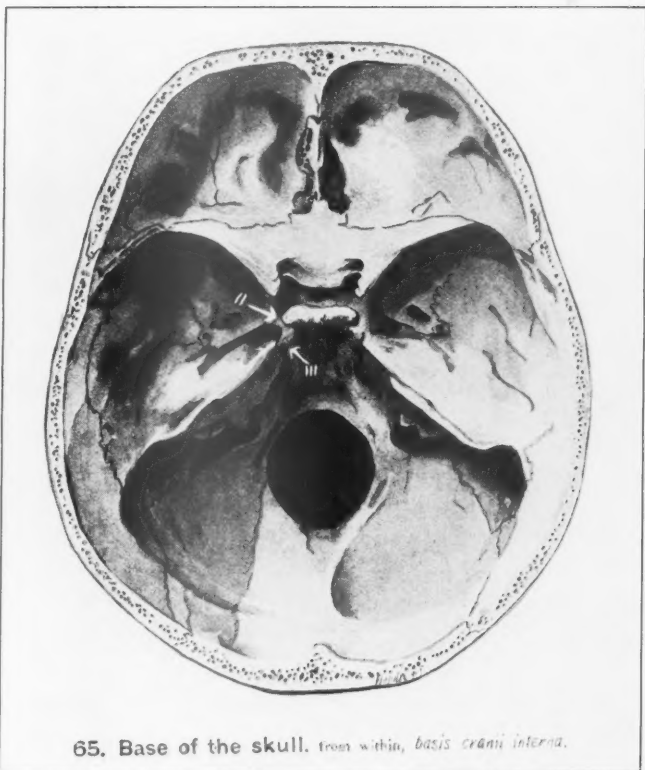


PLATE III.

From Spalteholz.

Arrow I—Sphenoidal spine of the petrous bone.

Arrow II—Posterior clinoid process.

Arrow III—Dorello's canal.

Note the (a) bony circumference of the canal, almost a half circle; (b) the extremely large canal; (c) absence of the accessory posterior clinoid process.

A cellular temporal bone.

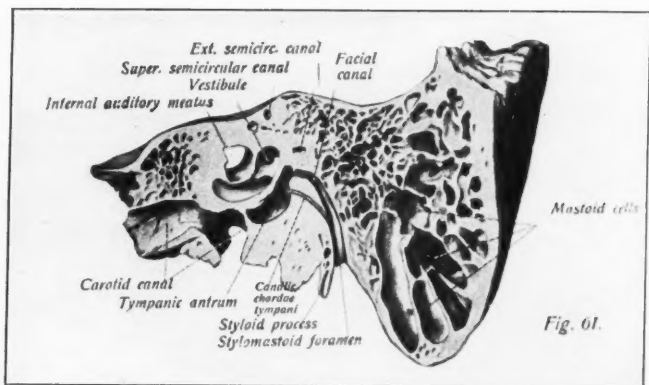


PLATE IV.

From Sobotta McMurrich.

Arrow I—Region of the trigeminal impression in which the Gasserian ganglion lies, and adjoining this the sphenoid spine, under which the sixth nerve is often placed.

Arrow II—Large cells underlying the above structures.

Note the extension of cells in all directions (a very favorable arrangement of tympanic suppuration).



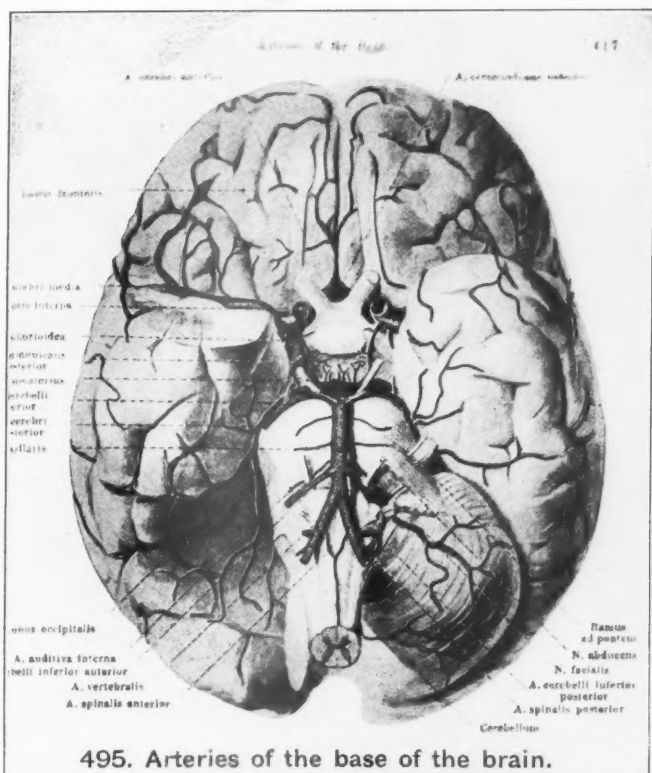


PLATE V.

From Spalteholz.

Showing the relation of the branches of the basilar artery to the sixth nerves. The sixth nerves are superficial to both branches of the A. Auditiva Interna, while both A. Cerebelli Inferior Anterior are superficial to the nerves just after they emerge from the brain stem. The position of the A. Auditiva Interna is generally superficial to the sixth nerves.

One of the illustrations from Dr. Harvey Cushing's paper "Strangulation of the Nervi Abducetes by Lateral Branches of the Basilar Artery in Cases of Brain Tumor."

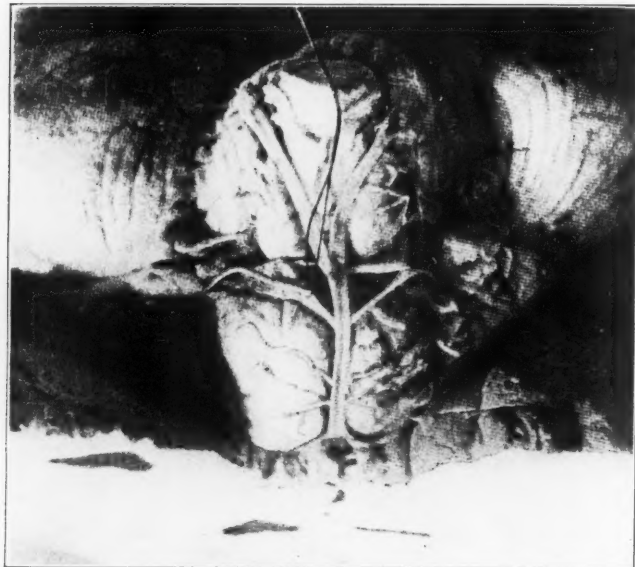


Fig. 9.

No. 13. Table 1.—Tuberculoma on left cerebellar hemisphere and multiple tuberculomas of cerebrum with edema and greatly increased tension. Fluctuating palsies of the sixth pair. Marked foraminal herniation.

NOTE: (1) Pons much grooved by all vessels, and even the abducens themselves have left imprints (cf. on right where nerve is turned out of bed.)

(2) On the left the two arteries arise from a single trunk which is superficial to the abducens and has deeply cut into pons and across the nerve (artery held away by black thread).

(3) The right abducens is also crossed and constricted by both A. auditiva int. and A. cerebelli inf. ant., which crosses the nerve just caudad to the pontomedullary groove (cf. figs. 10-13).

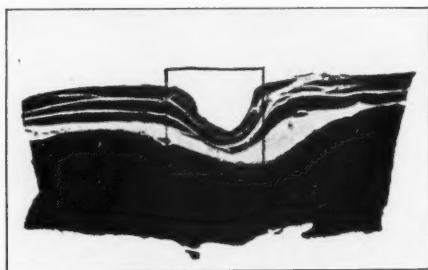


Fig. 10.

No. 13.—Longitudinal section of N. abducens sinistra showing imprint of A. cerebelli ant. inf. Squared area enlarged in fig. 11. Haematoxylin and eosin stain (cf. fig. 9).

PLATE VI.

From Brain, Vol. XXXIII, P. 204.

XX.

THE RADICAL CURE OF PERITONSILLAR  
ABSCESS.\*

BY HARRY L. BAUM, M. D.,

DENVER.

Most laryngologists will agree that the treatment of peritonsillar abscess is a disappointing experience, especially if the patient presents himself early in the progress of the disease. In view of the intense suffering involved and the many cases of serious and even fatal complications which have been reported, it is exceedingly desirable to afford relief as early as possible, rather than to wait until incision and evacuation are considered feasible. In spite of the fact that we may be certain of the diagnosis in the early or formative period of the disease, incision is usually without avail, or is at best only partially successful, and many authorities advise waiting arbitrarily until the fifth day before attempting to drain the abscess.

In order to meet these conditions, I have for some time been following the custom of removing the tonsil in cases of peritonsillar abscess, thus obtaining a radical cure most satisfying to me and to my patients. This procedure, although not new, has not yet received the recognition that it deserves, and it is the purpose of this paper to attempt to put it on a firm scientific foundation in the hope that its general adoption may result in the prevention of much needless suffering in the future. The method is particularly adapted to the relief of what I shall term second stage cases of the disease. In the late, or third stage cases, the abscess is ready to rupture, and relief can be promptly obtained in the usual way. In fact, the late case will usually relieve itself in a few hours, and I have known patients, subject to repeated attacks of quinsy, who had learned

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\*Read before the Mid-Western Section meeting, American Laryngological, Rhinological and Otological Society, Omaha, Neb., February 11, 1926.

that time was the essential element in the routine treatment of the disease and did not seek relief at the hands of the surgeon, knowing that his efforts would shorten their sufferings but little. Such a state of affairs is not one of which we can be justly proud.

At the last meeting of the Section I called your attention to certain anatomic relations of the tonsil,\* which I shall here briefly review as having a direct bearing upon this subject. The tonsil, with its thin fibrous investiture, lies in a bed formed by the superior constrictor muscle of the pharynx externally, the palatoglossus muscle anteriorly and the palatopharyngeus muscle posteriorly. Above, the upper pole of the tonsil extends into a space in the palate containing only loose areolar tissue and mucous glands, from which it is separated by its own fibrous investiture only. The tonsillar fossa is lined with an aponeurotic layer, a portion of the pharyngeal aponeurosis, which separates the tonsil from the superior constrictor muscle. Below, the tonsil blends with the lymphoid tissue of the base of the tongue, and its fibrous tissue capsule at this point becomes thicker and merges with the fibrous tissue of the pharynx. The line of cleavage is here not so well defined as at other locations in the fossa.

Elsewhere the tonsil is attached to the aponeurotic lining of the fossa by a comparatively few fibers of loose areolar tissue, except as the upper pole is approached, where the gland inclines inward and forward and is no longer in close apposition with the aponeurosis, which continues upward with the superior constrictor to its attachment at the base of the skull, sending but a few very delicate fibers over the upper pole of the tonsil. The upper portion of the tonsillar fossa, that part really extending into the soft palate, is thus left without an aponeurotic lining, except that of the palatopharyngeus muscle posteriorly and the palatoglossus muscle anteriorly. The latter muscle, however, occupies but a small part of the lower border of the palate, curving over in that border to travel downward to the tongue. Thus it is that the areolar space described ex-

\*The Mucous Glands of the Palate in Relation to the Upper Pole of the Tonsil. *ANNALS OF OTOLGY, RHINOLOGY, AND LARYNGOLOGY*, Vol. XXXV, No. 1

tends upward into the palate to a point above the palatoglossus muscle where there is nothing separating it from the oral cavity but the mucous membrane of the palate. Pus, migrating from the tonsillar fossa proper as a result of its expansion in a restricted space, follows the line of least resistance upward along the aponeurosis of the pharyngeal constrictor until it reaches the upper pole of the tonsil, and here finds less resistance to its expansion in the areolar tissue of the palate than at any other location in the region. Localizing here, it pushes forward through the palatal mucosa, the thinnest and least resistant place, just above the edge of the palatoglossus muscle. Sometimes, however, it burrows downward and forward under the palatoglossus muscle and opens into the supratonsillar fossa; and occasionally it ruptures through a crypt, usually in the upper pole of the tonsil.

Infection, penetrating the lining of the bottom of a crypt, migrates through the tonsillar capsule and becomes active in the potential space between the tonsil and the aponeurotic lining of the fossa. This, which I shall term the first stage of peritonsillar abscess formation, may occur at any point on the capsular surface of the tonsil, usually near or above the center. The activity of the infection rapidly results in the formation of pus, and the second stage of peritonsillar abscess formation is established. It is at this time, in my opinion, that the patient first feels the definite signs of the disease. The surgeon, on examination, finds the tonsil pushed toward the midline with the beginning edema and dusky congestion of the mucosa which is characteristic of the condition. The typical bulging above the tonsil is not yet present, although the pain is unmistakable and the dysphagia is marked. During this second stage of peritonsillar abscess formation ordinary methods of approach will not evacuate the pus, and it is therefore necessary to wait until expansion of the abscess in this limited space has resulted in migration upward around the upper pole of the tonsil in the areolar space in the palate. This, the third stage in the development of the disease, usually takes place in the space of three to five days, and may take longer in old cases with much peritonsillar fibrosis. Even at this stage it is not always possible to reach the center of the abscess with certainty, as old scars may so distort normal relations that the

pus migrates in various directions. In such cases it may follow the aponeurosis still farther upward to a point in the nasopharynx, or burrow through the aponeurosis and constrictor into the neck; extension downward into the mediastinum having been reported in a few rare instances. I have, in fact, found it necessary to dissect the tonsil partially from its bed in certain cases in order to evacuate the abscess, and such a method has been suggested by certain authors as feasible in these cases. This method of approach will be successful in a large majority of cases, but it is really doing that which I here advocate without finishing the task, for it is often necessary to dissect so far and so deep to reach the pus that the additional surgery incident to complete tonsillectomy adds nothing to the risk and very little to the extent of the operation.

From these observations we must conclude that at the inception of the second stage of peritonsillar abscess formation we actually have pus lying between the tonsil and the superior constrictor muscle. Clinically, this would coincide with the patient's early observation of pain and dysphagia and with the appearance of the throat described above, and precedes the occurrence of bulging in the palate. As soon as the surgeon is convinced of the diagnosis of second stage quinsy, as outlined above, I am of the opinion that drainage is indicated, and to secure that drainage tonsillectomy is necessary and is advisable. At operation, the abscess will be found to occupy a considerable portion of the fossa, usually extending upward toward the palate. The size of the cavity and the amount of pus it contains at this stage is surprising and immediately convincing of the advisability of the procedure. Inspection of the fossa after removal of the tonsil reveals an extensive infiltration beyond which a careful dissection will not extend, therefore not invading virgin soil for the possible extension of infection. However, I believe that the massive drainage secured by the removal of what really constitutes half the abscess wall practically obviates the danger of extension into uninfected tissue, and this opinion is fully borne out by experience and observation. In addition to this factor of safety, it is logical to assume that both local and systemic immunity must have reached a point of safety by the time definite suppuration has taken place.

The most gratifying aspect of my experience with this procedure has been the complete relief which results for the patient. These are the only adult tonsil cases in my practice not complaining of sore throat after operation; the ordinary post-tonsillectomy soreness is to them a pleasure compared with the suffering incident to quinsy. The temperature subsides immediately, the patient feels as well as the routine tonsillectomy, and, without exception, I find them deeply grateful for the relief afforded. The practice of this method of treatment has made me welcome the quinsy sufferer instead of dreading him, for not only is his suffering promptly and completely relieved but a needed tonsillectomy is also done. I may add that I remove the normal tonsil at the same time and without apparent ill effect of any kind.

It is my custom to use ether routinely for this procedure, as local anesthesia is both unsafe and inefficient. With careful suction the respiratory tract can be safeguarded against harm from aspiration of the pus. For lack of time and space I shall not attempt to anticipate and reply to the numerous objections which I expect these suggestions to arouse, but will leave them for the discussion.

In summary, my conclusions follow:

1. Anatomic and pathologic study and clinical observation have convinced me that pus is present in peritonsillar abscess long before it can be evacuated in the customary way.
2. Tonsillectomy evacuates this pus and provides massive drainage of the infected area.
3. By the time pus has formed, local infiltration and systemic immunity make tonsillectomy for drainage safer than futile incision or waiting.
4. The operation gives immediate relief from suffering and, at the same time, performs the additional service of removing the offending tonsil.

264 METROPOLITAN BUILDING.

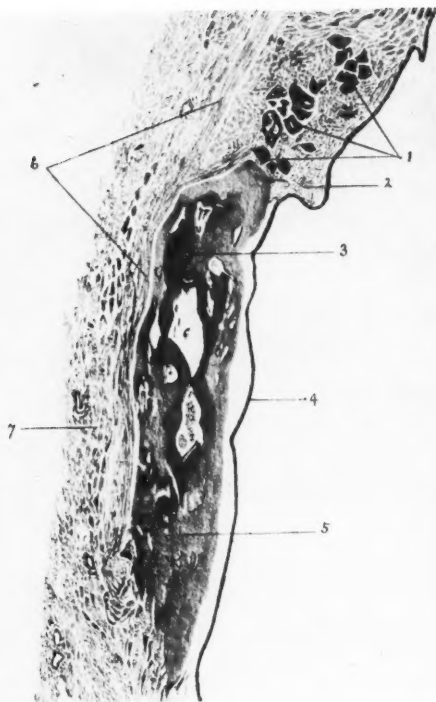


Appearance of tonsillar fossa immediately following tonsillectomy in quinsy case of two days duration. Central dark area of granulations represents outer or pharyngeal wall of abscess forming deep furrow extending upward into supratonsillar space above upper pole. Left fossa normal.





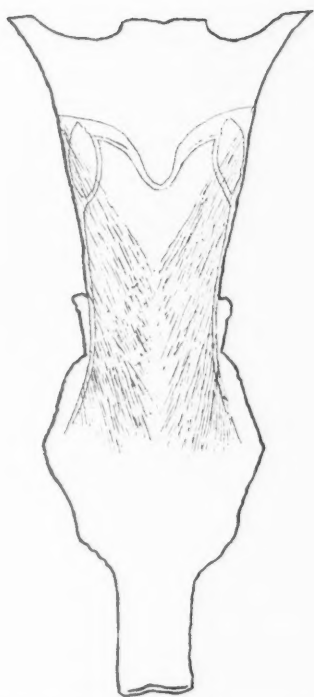
Tonsil removed from case of quinsy of three days duration. Central raised area is mass of granulation tissue representing probable original site of invasion. Surrounding area of lighter granulations represents abscess wall at time of operation showing extension upward over upper pole into palatal space.



Longitudinal Section Through Tonsil, Lower Palate and Pharynx.

1. Mucous glands of palate. Observe close relation to upper pole of tonsil.
2. Palatoglossus muscle, cut transversely in palate before turning downward in anterior pillar.
3. Tonsil.
4. Mucous membrane.
5. Fibrous tissue investiture of tonsil. Note increased thickness at lower pole, where it blends with the fibrous tissue of the pharynx.
6. Aponeurosis of superior constrictor muscle. Above it sends only a few fibres around the upper pole to be lost in the connective tissue of the palate.
7. Pharyngeal musculature.

NOTE: The tonsil is separated from its muscular bed by the pharyngeal aponeurosis (6). (Line of cleavage between aponeurosis and tonsillar capsule has been accentuated in drawing for purpose of illustration.) Infection passing from large central crypt of tonsil into peritonsillar space between capsule and aponeurosis and there forming pus would result in migration upward of pus along line of least resistance into palate. Migration downward is prevented by blending of fibrous tissue of lower pole with that of the pharynx.



Schematic drawing showing relation of tonsils to palate and pharyngeal aponeurosis. (Idea from Piersol, anatomic details from author's dissection.)

## XXI.

### MARBLE IN THE ESOPHAGUS. REPORT OF CASE.

BY E. LEE MYERS, M. D., AND U. S. SHORT, M. D.,

ST. LOUIS.

In reviewing the literature of approximately 1,800 cases of foreign bodies in the air and food passages, it was strikingly apparent that these intruders are seldom to be seen.

Mention of two cases similar to the one about to be reported was found. One,<sup>1</sup> a marble in the esophagus of a four-months-old child, located just above the cricopharyngeus. In this case Jackson used no anesthetic, Jackson's safety pin closer being used as a mechanical spoon in the removal, which was effected in four minutes and fifty-one seconds, by the oral method.

The other case cited by Jackson<sup>2</sup> was Dr. M. A. Goldstein's case of a marble in the air passages of a 10-year-old boy. An X-ray picture showed the marble to be in the right bronchus. The Jackson tube was passed and the usual grasping forceps were employed. The marble was about a centimeter in diameter, and it was impossible of removal with any of the usual instruments. Goldstein then resorted to low tracheotomy. The object failed to budge. He then passed a bronchoscope into the right bronchus and had the patient put into the Trendelenburg position. With good illumination he was able to see the foreign body, in fact, to touch it with a probe, and, turning the probe at almost a right angle, he was able to get beyond the marble. With the aid of the Trendelenburg position he teased the marble down into the trachea, and it was expelled with quite a quantity of fluid. Uneventful recovery took place.

In rehearsing the mechanics of the removal of the marble in this case, it was found that in addition to the mechanical spoon, Irwin Moore's tooth plate forceps would hold a marble securely enough to take it out of a rather constricted orifice, such as a Hubbard manikin, and also would remove it from the clenched fist.

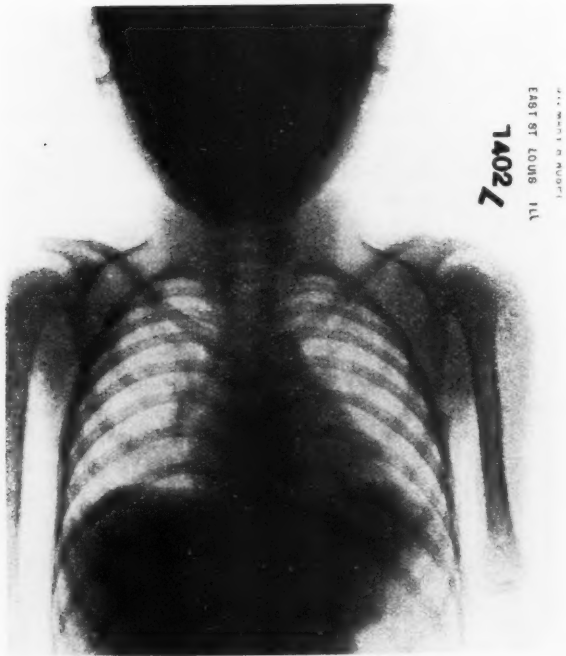
Naturally enough, a forceps would have to be placed beyond the poles of the globe, and this would be hazardous in the esophagus, as the folds may become entangled in the grasp of the forceps.

Case report, private service Dr. U. S. Short. Five-year-old child while at play gulped a marble; immediately complained of pain. Could not drink or eat, but was not dyspneic. An X-ray picture showed the marble to be at the level of the seventh cervical vertebra, approximately at the cricopharyngeus. An anteroposterior picture placed it distinctly to the left. The case was referred by Dr. Boyne to St. Mary's Hospital (East St. Louis) for esophagoscopy removal. Patient not anesthetized. Head holder, Dr. E. Lee Myers. Upon examination with a child's laryngoscope, no foreign body was seen. The same results were had with the child's esophageal speculum. Using a 7 mm. full lumen esophagoscope, a hard mass was felt in a clonic spasm. At this point considerable care was taken so that pressure on the trachea would not be made. After a few minutes' manipulation a click was heard as the child grunted, and the esophagus had cleared itself. Immediately the child could swallow water, and a radiogram showed the foreign body to be resting serenely in the stomach. The little one made a complete recovery. Bismuth was given as a precautionary measure to prevent an esophagitis.

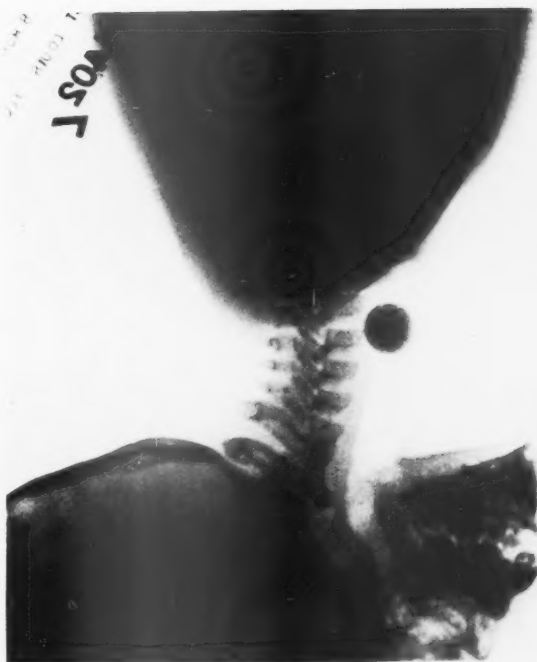
#### BIBLIOGRAPHY.

1. Annals of Otology, Rhinology and Laryngology, Vol. XXXIII (December, 1924), page 1144.
2. Discussion by M. A. Goldstein on Foreign Bodies in the Trachea. Proc. A. L., R. and O., 1913, page 388.

WALL BLDG.; METROPOLITAN BLDG.



Dr. Short's Case: Antero-posterior view showing marble in left crico-pharyngeal situation.



Dr. Short's Case: Lateral view of marble in crico-pharyngeus.

## XXII.

### A SKIN-PERIOSTEAL FLAP FOR THE RADICAL MASTOID.

BY D. CAMPBELL SMYTH, M. D.,

BOSTON.

Since the completion of this paper containing therein the report of work which I considered to be original, I find that a very similar skin periosteal-flap was done a number of years ago by Dr. E. M. Holmes and reported at a meeting of the Eastern Section of this society in 1907. Although it was very disappointing to me to find that the similar piece of work had already been done, I am glad that I discovered it before the paper was published, and take pleasure in giving Dr. Holmes the credit to which he is due. I shall therefore change the title, striking out the "new" and thus entitling my paper "A Skin-Periosteal Flap for the Mastoid." I consider it worth while to report this procedure because the technic for procuring a flap is different to that described by Dr. Holmes, and also because I have drawings and lantern slides to demonstrate that technic, and lastly because I have a number of cases operated on, the final outcome of which I shall present.

The process of epidermization of a radical mastoid cavity starts from the skin of the anterior wall of the auditory meatus which is left intact and from any other area of epidermis which may be implanted in the cavity, either by plastic flaps or grafting.

The object of the so-called plastic operations on the external auditory canal, after the complete exenteration of the mastoid, is to prevent a stricture of the external meatus and to utilize the posterior wall of the meatus for partially covering the surface of the wound in the bone, thus adding another starting point for the epidermization of the uncovered granulating bony surface.



The first plastic flap proposed by Stacke has undergone many changes and modifications. Stacke took the skin and subcutaneous tissues over the mastoid process and endeavored to implant them in the bony cavity. This procedure has the disadvantage of leaving an open wound, which is usually sloughy behind the ear. Panse in his flap incised the posterior wall of the meatus in a direction parallel to its bony axis as far as the auricle. At the external extremity of this incision in the auricle two right angle incisions were made for the purpose of forming a superior and an inferior flap. The Körner flap, with which we are all so familiar, at least in Boston, is a modification of a method originally suggested by Panse for those cases in which the posterior wound has to be closed by primary union. Körner prolonged the two parallel incisions into the concha, whereas Panse's reached only as far as the concha.

Brühl used a flap consisting of a combined Panse and Körner. Other flaps by otologists, such as Ballance, Passow, Siebenmann, Whiting, Lake, Bryant, and so forth, have been used with success. Of them all, probably the most simple and most universally used is that of Körner. The Siebenmann flap gives a very large external meatus, sometimes so large, I think, as to be disfiguring.

Back in 1909, 1910 and 1911, Mosher tried many methods of utilizing the posterior subcutaneous tissues of the mastoid, somewhat after the method of Stacke with modifications. His endeavor was to use those tissues to obliterate, or at least make smaller, the bowl of the cavity. Being house officer at the Infirmary at that time, I assisted him on many of these cases. From this work the idea came to me which has resulted in the flap operation I am about to describe to you. His work gave me the idea that our plastic work might be very much improved and the tissues of the ear and about it better utilized to help in filling and promoting healing of the radical cavity.

My experience, and I think the experience of most men, has been that after the radical operative procedure is done the posterior canal skin was so traumatized that the Körner flap cut therefrom is practically useless. The very thin end of it, which came from the region of the annulus, usually had to

be cut off, if indeed it had not already been torn free in its removal. The procedure, the technic of which I am about to describe, is, therefore, not intended to supplant the skin graft but simply to improve the skin flap, by implanting live epidermis with a blood supply through its attached periosteum deep into the recesses of the radical cavity.

I first performed the operation on a wet head in Mosher's laboratory at the Harvard Medical School in 1912. The flap came out all right, as I had expected, but it required more than an hour to dissect the required tissues free and then with the greatest of difficulty. It never occurred to me that periosteum separates up much more easily on the living subject. So thinking that a flap which took such a long time to procure was useless, I placed the specimen back in the jar with no hope of ever trying the procedure on the living. Not until six years later, when I mentioned and described the procedure to Dr. Walker during our hospital service in 1920, did I use it on the living, and then only at his urging. I am pleased to say that once the technic is acquired, the time required for procuring the flap is usually not much more than five to ten minutes.

#### LANTERN SLIDES.

The technic of the procedure of cutting the skin flap is as follows: After the ear has been prepared in the regular manner for operation, a gauze wick saturated with 7 per cent tincture of iodine is placed in the canal for about half a minute; this is removed and followed by a gauze wick saturated with alcohol, for about a minute. The canal is then thoroughly dried out and the primary incision made. This incision is shown in drawing I.

A canal knife is introduced along the superior canal wall to the promontory, and an incision is carried outward along the superior canal wall to the external orifice; this cut must be carried through the periosteum to the bone; the knife is then turned over and the same procedure is followed on the inferior canal wall, again making sure that the incision is carried through the periosteum right down to the bone. The canal is now temporarily packed with gauze and left for the time be-

ing; the regular mastoid incision is now made down to the periosteum, but not through it; the subcutaneous tissues are then dissected forward, the ear being held forward with some tension by the assistant; as the tissues are dissected forward, the posterior cartilaginous canal will come into view, as shown in drawing II; at the junction of the cartilaginous and bony canal the knife is carried directly through, as shown again in drawing II; this incision will meet and intersect the original incisions, already described, in the external canal; from the two points of intersection of these incisions, as shown in drawing III, incisions are carried backward through the periosteum over the mastoid to the edge of the posterior incision. With an ordinary submucous elevator the periosteum of the mastoid is easily undermined, working from these incisions toward the center. Next, directly back of the bony canal, which now shows up plainly in the field of operation, a small buttonhole is made in the periosteum; now introducing a sharp septum dissector through this buttonhole and following down under the skin of the posterior canal wall, it is easily lifted from the underlying bone; the only point at which difficulty may be experienced for a short time is directly over the spine, where the tissues are very adherent; having freed up the skin of the posterior canal the whole flap consisting of the periosteum over the mastoid, having at its distal end the skin of the posterior bony canal, now easily lifts out and backward, as shown in drawing IV, giving us a wonderful view of our operative field before us. Having now covered the flap with gauze saturated in warm saline solution, an ordinary retractor is introduced in the ordinary way (no special retractors are required, as no skin flap has to be held out of the way). I wish to lay stress here on the point that, in direct variance to all other flaps, in this procedure the whole plastic flap is posterior to the operative field. The field is now ready to do the radical, with an excellent view of the landmarks and no bleeding outside of that from the bone to bother. (The radical is completed, as shown in drawing V; in this drawing I have tried to bring out what I consider an important point, namely, that the posterior rim of the cavity must be rounded to a nicety.) The reason for this is that a sharp edge at this point might easily shut off blood supply through the perios-

teum when it is laid into the cavity and packed into place, as shown in drawing VI. The skin flap usually is placed so that the tip of it reaches just short of the aditus, and it may be necessary to make short cuts through the periosteal flap at its upper posterior attachments, in order to have it lie nicely in place in the bowl of the cavity. The next procedure, and one which I regard as one of the most important steps in the technic, is the splitting outward of the cartilaginous canal wall through the concha, as seen in drawing VI. It is not necessary to remove the cartilage, for having carried the incision the necessary distance, especially the lower incision, through the concha so that the little finger may be easily thrust through the canal, several No. 1 catgut sutures are introduced into the subcutaneous tissues of the concha and posterior lip of the mastoid wound and tied. These sutures hold the canal widely open. My experience has been that this piece of cartilaginous canal and concha which is thus tied back into place is about the size and as useful as the Körner flap usually employed. Now, picturing the cavity diagrammatically, it has several areas from which it can epidermatize. First, there is the anterior canal wall, the skin of which has not been touched; second, looking at the cavity posteriorly from without inwards, there is the skin of the cartilaginous canal and concha, which is anchored back in place, taking the place of old Körner; next comes an area of the bowl covered by periosteum, and internal to that the epidermis of the posterior bony canal, nourished by a blood supply through the periosteum. In my first cases the operative procedure ended here. The tissues were gently packed into place with weak iodoformed gauze, which was allowed to remain in situ for a week and then removed. My procedure, after removal of the gauze, was to have the cavity filled twice a day with sterile vaselin expressed from a tube directly into the cavity. In those cases there was, as I expected, more or less of a battle with granulations in the middle ear, but eventually the cases did well, resulting in perfectly dry ears, although taking a little longer time than following a complete skin graft. I have eliminated my middle ear trouble on the last services at the Massachusetts Eye and Ear Infirmary by introducing, at the time of the operation, a small skin graft. The only reason I rather hesitated in putting a skin graft in the

middle ear at first was because it would be necessary to take the packing out on the third or fourth day, and I was somewhat afraid of a collapse of my canal and flap, but in cases which I have the canal has stayed wide open and the periosteum and skin flap have remained tightly glued to the bone. One of the things I feared most in trying this new procedure was the postoperative sloughing of my periosteal flap. I am pleased to say that not in one of the cases done has there been any sign of sloughing or pus in the cavity following the operation.

The sterile vaselin as a dressing has worked so well that I still continue to use it. After the packing has been removed from the cavity, nothing at all, not even a dressing forceps or a cotton swab, is introduced. Silver nitrate is never used. A nasal speculum is used to inspect the cavity daily, and whenever a granulation appears it is effectually and speedily removed by a large Greenwald ethmoid punch. After epidermization of the facial ridge and bowl is complete, and perhaps only a small area remains in some part of the middle ear, I do not hesitate to use cotton swabs freely in the cavity. In private cases which can be well kept under supervision, I am inclined to think that the auxillary skin graft in the middle is unnecessary, but in hospital cases, where the patient sometimes gets away from supervision, as two of mine did, it is a great aid.

As regards the results, so far all the cases which have been done, varying from six months to six years ago, are dry and have no moisture in the middle ear. In not one of the cases operated on was there any pus discharge from the cavity, using the treatment outlined above. Two of the cases which I did without a skin graft in the middle ear got away from my supervision within six weeks of the operation. They had no further treatment. When they appeared at my office some months later, they had dry middle ears. I think those cases well exemplify what Nature will do if the operation has been fairly complete. In regard to the later cases done with a primary skin graft in the middle ear, the time of complete epidermization of the cavity was short, averaging between three and four weeks, so that I should say that they averaged up in this respect better than the cases in which I used to do a complete

skin graft with a Körner flap. My experience with this latter procedure was that some of the cases had a remarkably short convalescence, while others took months before complete epidermization took place. In all of the cases the posterior wound healed by first intention, so that the resulting scar cannot be seen in most of them.

Advantages of the Operation.—First, the flap is cut at the beginning of the operation and placed out of the way. Second, there is a dry operative field, with an absolutely unobstructed view of the posterior canal wall and the middle ear, even as good a view as we get of those structures in the extensive Lake incision, and all will agree with me that a good operative view in radical mastoid work is half of the operation. Third, absolutely untraumatized tissues are introduced into our mastoid cavity to form the plastic flap, rendering the liability to sloughing and infection in the cavity much less. Fourth, the periosteum helps to diminish the size of the bowl, but in none of the cases, some of them done five years ago, has there been any tendency to the formation of excessive bone in this region. Fifth, all of the tissues being back of the retractor, a skin graft is easily placed with accuracy in the middle ear. Sixth, the time of operation is shortened, if anything; at any rate, it is not lengthened. The result, with this procedure as with any other, may be good or bad. A good result, whatever method of plastic or grafting is used, presupposes a good operation, and by a good operation I mean a complete one and, at the finish, a cavity perfectly smooth to the eye and touch.

#### CASES.

I am indebted to Dr. Mosher for the opportunities at the Harvard Medical School to work out this operation. To Dr. Walker, for giving me the opportunities and encouraging me to try this procedure in the first cases. To Mr. H. F. Aitken of the anatomic department at Harvard, for the excellent drawings made from wet specimens.

The following cases I am reporting briefly, and on all of these cases a complete neurologic examination, Wassermann, labyrinth, including rotation, caloric and fistula tests, and X-ray of the mastoid were done. A complete hearing test was

also made on all. In the short summary of the cases I shall simply give the diagnosis from the records, the date of the operation and a few short notes on the postoperative healing.

Case 1.—H. S., tailor, age 36, married, admitted March 4, 1918. Diagnosis: Chronic suppurative left middle ear with labyrinthitis and cholesteatoma. There was a past history of polyps being removed several times in the outpatient department up to December, 1920.

December 27. He was admitted at the Eye and Ear Hospital for observation and treatment. His chief complaint was dizziness. At that time his Wassermann was negative, and an X-ray showed cholesteatoma in the left mastoid.

January 6. Operation. Left radical mastoid with skin-periosteal flap.

January 13. All packing removed. Cavity filled with sterile vaselin.

January 30. Cavity looks well. Almost epidermatized. Nearly dry.

February 1. Patient discharged to outpatient department.

February 6. Doing well. To report to Dr. Smyth's office. This patient lived out of town and at this date disappeared. He never reported at the office until about a year afterward. The cavity at that time was completely epidermatized and dry, with a couple of small adhesions across the middle ear. These adhesions made no particular difference, as the patient had no hearing in the ear before operation.

Case 2.—H. B., auto machinist, age 28, married, admitted January 13, 1921. Diagnosis: Left chronic suppurative ear with vertigo. The fistula test was strongly positive. Violent twitching of the eyeballs with rotary nystagmus, producing considerable dizziness and nausea.

January 18. Operation. Left radical mastoid with skin-periosteal flap. Cholesteatoma found, also a fistula of the horizontal semicircular canal.

January 26. All packing removed, with cavity epidermatizing well. Slight edema back of the ear.

January 31. Patient discharged to outpatient department. Cavity epidermatizing well. Discharge very slight.

This patient was also lost sight of for a number of months. After going home he had an acute cardiac decompensation and was confined to bed for a number of weeks without any ear treatment. When he reported to my office a few weeks later the cavity was completely epidermatized with very few adhesions.

Case 3.—A. L., schoolgirl, age 10, admitted January 4, 1922. Diagnosis: Left chronic suppurating ear with polyps of eight years' duration. The history showed that polyps had been removed many times. When I saw her she had a large polyp almost filling the canal of the ear, although one had been removed a week previously.

January 18. Operation. Left radical mastoid with skin-periosteal flap. In this case a small skin graft was put in the middle ear.

January 23. Silver clips and packing removed. Postaural wound healing nicely. Middle ear cavity looks excellent. Graft has taken well. Cavity filled with sterile vaselin.

January 31. Patient discharged to the outpatient department. She was not in my office until March 20, 1922, when the cavity was all dry and epidermatized. This patient has been seen at intervals since that time and the ear has remained perfectly dry.

Case 4.—P. H., male, age 53, admitted January 7, 1922. Diagnosis: Double chronic suppurating ears with dead labyrinth and cholesteatoma in both mastoids.

January 17. Operation. Right radical mastoid with primary skin graft and Smyth flap. Small skin graft was placed in the right middle ear after the operation.

January 23. Silver clips removed from posterior wound, also all packing removed.

January 31. Cavity epidermatizing well. No pus. Discharged from hospital.

The middle ear of this patient did not get completely epidermatized until December 15, 1922. He has been seen at intervals since and has remained perfectly dry.



Case 5.—A. J., age 13, admitted December 14, 1923. Diagnosis: Chronic suppurating otitis media. Patient has been a frequent patient in the hospital since August 9, 1921, for treatment of chronic suppurating ears. Tonsils and adenoid have been removed and polyps at various times.

December 18. Operation. Left radical mastoid with primary skin graft and Smyth flap.

December 24. All packing removed from cavity. Skin clips removed from posterior wound, which has healed by first intention. Mastoid cavity filled twice a day with sterile vaselin.

December 31. Middle ear epidermatizing, discharge very slight.

January 9. Discharged to the outpatient department.

February 6. Patient got complete epidermatization, with a dry cavity, except for a small area in the posterior part of the floor of the middle ear. This area was about a couple of millimeters in diameter and produced a slight secretion in the floor of the middle ear until September of the same year.

Case 6.—T. W., female, age 9, admitted to hospital November 12, 1923. Diagnosis: Right chronic suppurating otitis media with cholesteatoma.

November 15. Operation. Right radical mastoid with skin-periosteal flap and skin graft in middle ear.

November 19. Packing removed from cavity, also skin clips removed. Cavity to be filled twice a day with sterile vaselin.

November 28. Cavity epidermatizing well. Patient discharged to the outpatient department.

December 22. Under nitrous oxid gas granulations were removed from the bowl of the cavity. No further trouble was experienced with granulations in this case. The cavity went along to complete epidermatization in the normal time. Patient now has a dry middle ear and was last seen October 21, 1924.

Case 7.—G. B., age 10, admitted January 21, 1924. Diagnosis: Left chronic suppurating otitis media with dizzy spells for the past three months, these spells being accompanied by nausea and vomiting.

January 23. Operation. Left radical mastoid with skin-periosteal flap and skin graft in the left ear.

January 29. All packs removed from the cavity. Skin clips removed from the wound. Vaseline in cavity twice a day.

Patient kept in hospital until February 22nd, because of rather persistent foul discharge. Cavity meanwhile epidermatized very nicely without any granulations. This patient was last seen October 1, 1924. Cavity is completely epidermatized.

The remaining three cases I am not reporting in detail were done by other men whom I assisted cutting the flap. I understand from them that the final result was entirely satisfactory.



Fig. 1.

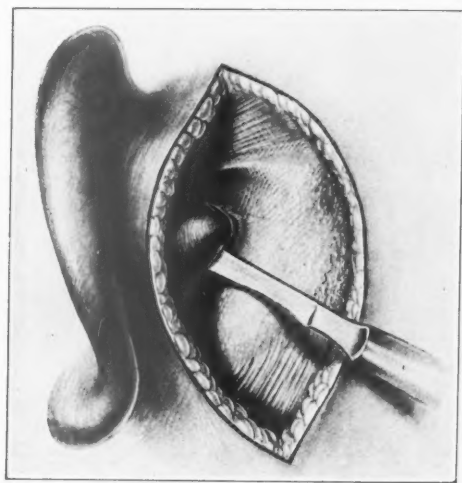


Fig. 2.

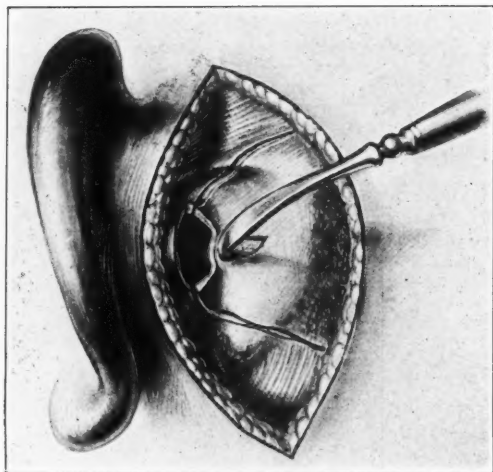


Fig. 3.

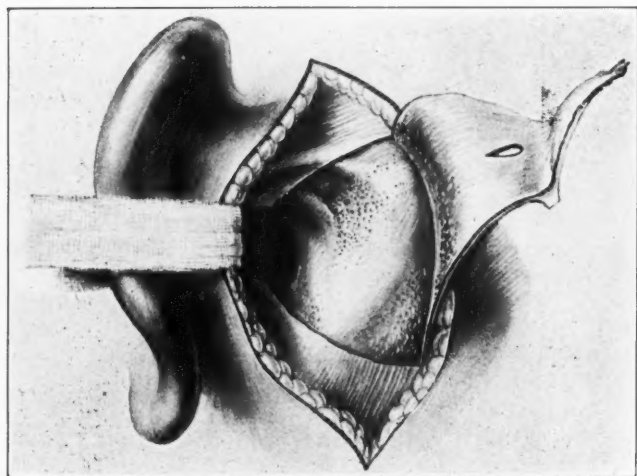


Fig. 4.

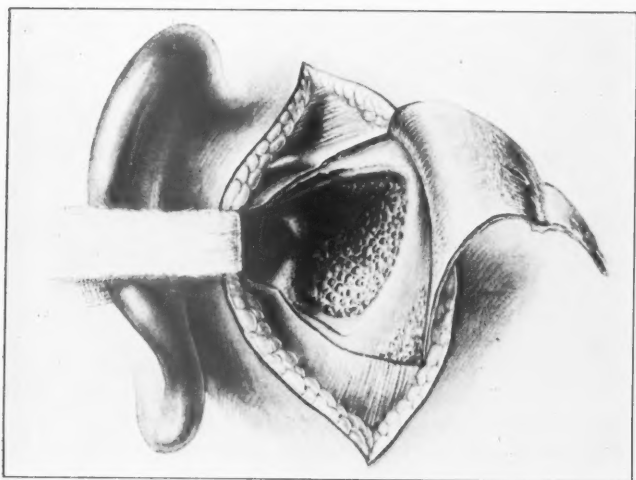


Fig. 5.

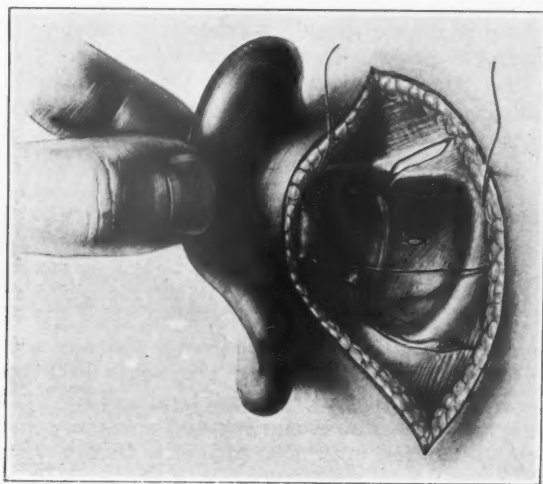


Fig. 6.

XXIII.

THE EARLY HISTORY OF THE AMERICAN OTOL-  
GICAL SOCIETY WITH SPECIAL REFER-  
ENCE TO ITS FOUNDERS.\*

BY THOMAS J. HARRIS, M. D.,

NEW YORK.

Two years from next month this Society will celebrate the sixtieth anniversary of its existence.

The near approach of this event, which has had so much to do with the development of otology in this country, makes this a fitting occasion to consider the early history of the Society and to recall those pioneers whose keen scientific zeal and discerning wisdom led to its foundation.

The growth of otology in the United States was exceedingly slow in the eighteenth and early part of the nineteenth centuries. This was only natural, in as much as Europe showed little progress in the specialty during this period. The two outstanding contributions of the eighteenth century were the first catheterization and injection of the eustachian tube in 1724 by Guyot, the postmaster of Versailles, and the first trephining of the mastoid process by Petit in 1750, who used a gauge and hammer. He was followed by the army surgeon Jasser in 1776.

The first book on the ear was published in this country in 1821. It was the American edition of the "Anatomy of the Human Ear," by John Cunningham Saunders, an Englishman, edited by William Price, M. D., of Philadelphia. This was a brief but scientific treatise, deficient in all reference to any method of examination of the drum membrane. Price was a surgeon to the Pennsylvania Hospital. Saunders' book was followed by the translation from the French in 1829, by Nathan R. Smith, a distinguished surgeon of Baltimore, of Saissy's book on the "Diseases of the Internal Ear." Smith was not an

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\*President's address, fifty-ninth annual meeting of the American Otolological Society, Montreal, June 2, 1926.

aurist, but added twenty pages on the external ear, in which he called attention to the use and value of the catheter and recommended the method of Kramer of examining the drum membrane by direct sunlight after straightening the canal. In 1864, D. B. St. John Roosa made a translation of von Trötsch's "Diseases of the Ear." Virtually down to the time of the discovery of the laryngoscope, in 1855, the practice of otology was chiefly in the hands of quacks. The late Stanton A. Friedberg, in the "Annals of Medical History," Volume I, page 86, writes entertainingly in an article on "Laryngology and Otology in Colonial Times," of a country doctor by the name of M. Wilson, who lived near Philadelphia in the latter half of the eighteenth century and who compiled a book called "A Therapeutic Alphabet." In it he dwells at some length on the prescriptions for ear troubles of a certain "Dr." James Graham, who was perhaps the earliest ear quack in America, flourishing in Philadelphia about 1773, commenting upon them in a considerable spirit of combativeness and antagonism—we quote:

"Dr. Graham ye ōtistis rules by which he pretended to cure inveterate deafness were these:

1. Bleed the jugular 13 oz. every 10 days for three times.
2. Three emetic Boluses, given one ye day after each bleeding.
3. A mixture night and morning (perhaps tincture sacra & amara mixture) drinking sage, sasafras and Fennel seeds, Tea.
4. His accoustic essence (is?) in each ear and yn wt Force s(n)uffing it up ye nostrils as long. But juice of ground ivy—rue—rosemay & garlic 2 oz. in hot tar water would be perhaps better."

Graham's advertisement added that those needing his services are advised to take advantage of them immediately, as he is about to sail for England.

Roosa, in the first edition of his treatise on "Diseases of the Ear," which appeared in 1873, throws an interesting light on the condition of otology in the years immediately preceding, as follows:

"In the preface of a translation of 'von Trötsch on the Ear,' published a little more than nine years since, the trans-

lator had so little faith in a general professional interest in the diagnosis and treatment of diseases of the ear that he quoted a proverb to indicate that an ordinary human life would not suffice to see the fruit of the tree then being planted, in presenting to the English speaking profession a work which has done much for the progress of otology.

In view, however, of the active and permanent interest in this subject, which has shown itself in the formation of societies, the establishment of journals, improvements in methods of practice and a general appreciation of diseases of the ear, the author can but felicitate himself that even in a short life he has seen some fruit of a tree which, although he did not plant, he at least assisted in cultivating.

The practice of otology in this country was, a few years since, almost exclusively confined to charlatans; but it is now cultivated by a class of men who are the equals of any in the profession. Ten years ago, in most parts of the country, those who wished advice upon a disease of the ear were forced to seek aid outside of the profession. At the present time there can be found those in the large cities who are constantly and successfully treating aural diseases; and all over the land the old and familiar advice, "not to meddle with the ear," is growing far less frequent. The day will soon arrive—if indeed it be not already upon us—when otology will take equal rank with ophthalmology, to which department it has so long been a mere appendage, and when some knowledge of the diseases and treatment of the ear will be required of every practitioner."

To this wholesale indictment of scientific otology previous to the '60's, a few conspicuous exceptions must be made. One of these is Dr. Joshua L. Cohen, of Baltimore. Reeves' "American Edition of Cooper's Dictionary of Practical Surgery," 1846, speaks of him as follows:

"In the United States there have been few surgeons who have distinguished themselves by their success in the treatment of diseases of the ear. Deafness is a less frequent disease than in Europe, but cases are nevertheless sufficiently numerous to deprive us of any justifiable excuse for the neglect which this subject has received from American surgeons.

"Dr. Cohen of Baltimore and Dr. Dix of Boston have for several years directed their particular attention to diseases of



the internal ear, and to the investigation of the abnormal conditions of the tympanum and eustachian tube. In cases of deafness, these gentlemen have employed condensing apparatus for administering the air douche through the eustachian tube, after the plan of Kramer and others. By the air and also by the water douche, these gentlemen have acquired great tact in the diagnosis and treatment of obstructions in the tube and upon the tympanum.

"In 1840, he established, in connection with his friend, Dr. L. Samuel Chew, an eye and ear institute in Baltimore, in which Dr. Chew had charge of the eye department. He wrote one paper describing 'Postmortem Appearances in a Case of Deafness.' The paper is very short but is written in a most scientific manner."

The discovery of the laryngoscope, in 1855, gave a tremendous impulse to the scientific study of the ear, and among the men graduating about this time from the various medical schools in this country there were a number who determined to perfect themselves in every way possible in this subject. Most of them went abroad to study, especially to Vienna, and there spent long periods of time in acquiring all that was known in otology. Sir William R. Wilde was the pioneer in modern otology in Great Britain, bringing out in 1853 his treatise on the ear entitled "Practical Observations on Aural Surgery." This was followed by Toynbee's classical book on "Diseases of the Ear," in 1860, and by von Tröltsch's "Anatomy of the Ear," the year following, 1861. In Vienna, Politzer, under whom many of us have studied, had just described his method of injecting air into the middle ear, and Schwartze in Halle had issued the first number of the *Archiv für Ohrenheilkunde*. With scarcely an exception, these young Americans were all interested in ophthalmology as well as otology, and they returned to their native country in the period at the close of the war to inaugurate a new era in the practice of these specialties. The American Ophthalmological Society was founded in 1864 (June 7).

It was the thought of the founders that the two subjects of ophthalmology and otology could be considered at the same meeting at separate sessions. The preface of the "Transactions of the American Otological Society," seventh annual meeting,

1874, states the reason for the separation and for the foundation of the American Otological Society.

"Newport, R. I., July 15, 1874.

"Until within a very few years, the science and art of otology had been almost entirely neglected by the medical profession of the United States. In this respect, however, we were not much behind most other parts of the civilized world.

"In its very best position, otology was an appendage, not always very gracefully worn, to the Department of Ophthalmology, for in this country, as in Ireland, diseases of the eye and ear have always been connected, both in the minds of the profession and the laity. The Ophthalmological Society of the city of New York thus has for its object the cultivation of both ophthalmic and aural science.

After the American Ophthalmological Society had proved itself a useful organization and had become firmly established, the question of amending its constitution so as to admit of the discussion of aural subjects, was considered by the members, nearly all of whom were engaged in the practice of both ophthalmology and otology. It was proposed to devote one day of the annual sessions of the society to aural medicine and surgery; but it was finally decided that such a union could not produce satisfactory results. Consequently, on the 22nd of July, 1868, the American Otological Society was organized by certain members of the American Ophthalmological Society who were then at Newport, Rhode Island, in attendance upon the fifth meeting of the latter. Their names will be found in the minutes of the first meeting, which are printed in this volume.

"As will be seen by reference to the record, no scientific business was then transacted, but at each subsequent annual meeting papers have been read and discussion held. It is believed that these articles and debates have contributed essentially to the interest in aural science that now obtains in this country and abroad; and it is confidently hoped that the Otological Society has but just begun a career which is to continue so long as medical science is cultivated."

This hope, we will all agree, has been abundantly fulfilled.

The minutes of the first meeting are brief and to the point:

## "THE AMERICAN OTOLOGICAL SOCIETY

Newport, R. I., July 22, 1868.

At the meeting for the formation of the American Otological Society, held July 22, 1868, at the Ocean House, Newport, R. I., the following gentlemen were present. viz.—

Drs. C. R. Agnew	New York
H. D. Noyes	New York
D. B. St. John Roosa	New York
F. J. Bumstead	New York
D. D. Pomeroy	New York
John Green	St. Louis
E. Williams	Cincinnati
C. A. Robertson	Albany
C. E. Rider	Rochester

Dr. E. Williams was elected chairman, and Dr. C. E. Rider secretary.

A Constitution and By-laws were adopted.

The following gentlemen were then elected members, viz.—

Drs. Samuel L. Frank	Baltimore
J. Orne Green	Boston
W. W. Morland	Boston
Charles E. Hackley	New York
R. F. Weir	New York
Francis Simrock	New York
W. S. Ludlum	New York
W. F. Holcombe	New York
A. D. Williams	Cincinnati
J. F. Noyes	Detroit
John H. Dix	Boston
E. L. Holmes	Chicago

Dr. E. Williams was elected president for the ensuing year, and Dr. C. E. Rider recording secretary and treasurer.

The president appointed Dr. D. B. St. J. Roosa the Committee on the Progress of Otology.

The secretary was directed to procure the publication of the Constitution and By-laws for the Society.

Adjourned to meet at the Ocean House, Newport, July 20, 1869, at 11 a. m.

C. E. RIDER, Secretary."

These show that nine men only were present at the initial meeting in Newport. Four of them were from New York, one from Cincinnati, one from Albany and one from Rochester. At this meeting, twelve men were elected, four from New York and three from Boston, making in all twenty-one members the first year.

Meetings have been held annually, with the exception of the year 1876, when, by vote of the Society, the scientific proceedings were transferred to the first International Otological Congress, which met in New York City, and in 1877, when a quorum was not present at Niagara Falls, on account of a railroad strike. There have been, in all, twenty-nine presidents of the Society and nine secretaries and treasurers. Eighteen volumes of the Society, covering fifty-six years, have appeared.

The first president of the society was Dr. E. Williams of Cincinnati. He was succeeded by Dr. Henry D. Noyes of New York, who served as president for four years.

There were no scientific proceedings of the first annual meeting, in 1868, and the proceedings of the second annual meeting, in 1869, were published with the proceedings of the American Ophthalmological Society. An error occurred in the numbering of the volumes, No. 10 unintentionally having been omitted.

The proceedings of the early years of the Society are represented to a large extent by the work of six or eight men—Roosa, Knapp, Buck, Sexton, of New York, Blake and Greene of Boston, and Burnett of Philadelphia. To this number should be added Agnew and Pomeroy, who were less conspicuous contributors.

Agnew, Roosa, Pomeroy and Greene were present or elected at the first meeting. Knapp was elected at the next meeting, Sexton at that meeting, and Burnett in 1872.

The little group of nine men which gathered at the Ocean House, Newport, on the 22d of July, 1868, elected Dr. E. Williams as their chairman, and later as president for the first year of the Society. Dr. Williams was considerably older than most of those who had an active part in the early work of the Society. He was born in 1822. After graduating in arts and in medicine in this country, he studied the newly developed specialty of ophthalmology abroad under von Graefe, settling

upon his return to America in Cincinnati. He contributed extensively to ophthalmologic literature, but no papers to the Otological Society. He died in 1888.

Another member of the little group was Dr. Cornelius R. Agnew, of New York. Dr. Agnew was born in 1830. He graduated from Columbia, class of 1849, and from the College of Physicians and Surgeons in 1852. In 1855 he was appointed surgeon at the New York Eye and Ear Infirmary. He was one of the four founders of the Union League Club of New York. In 1866 he established the ophthalmologic clinic in the College of Physicians and Surgeons. In 1868 he originated the Brooklyn Eye and Ear Hospital, and in 1869 the Manhattan Eye and Ear Hospital. He was a trustee of Columbia from 1874 to the time of his death. He was not a frequent contributor to otologic literature, but at the third annual meeting he read a paper reporting a case of mastoid disease successfully trephined. A one-half inch trephine was used. This was one of four cases operated upon with a gimlet in 1864 by Dr. Dixi Crosby of Hanover, New Hampshire. This is one of the earliest trephining operations performed in this country. Even at this early date the keen scientific mind of Dr. Agnew was led to state, "I am convinced that mastoid cell disease is very common in ear diseases, that it may escape attention and that we often lose time by not making an early opening."

The third distinguished member of this group was Dr. Henry D. Noyes of New York who, in 1864, with Dr. Derby of Boston and Dr. Baumstead of New York, was instrumental in organizing the American Ophthalmological Society. Dr. Noyes was for many years one of the leading ophthalmologists in New York City. He was for a long time surgeon at the New York Eye and Ear Infirmary. He was born in 1832. He graduated from the New York University, A. B., '51. He was president of the American Otological Society for four years following Dr. Williams. An extensive contributor to ophthalmologic literature, he contributed no paper to the proceedings of the Society.

Without doubt the most distinguished member of this group of founders, from an otologic standpoint, was Dr. D. B. St. John Roosa. Scarcely thirty years old at the time, he already had come to occupy a leading place among ophthalmologists

and otologists in America. He was small in stature, possessed of high intellectual powers, a sonorous voice and a commanding personality, and may be regarded as one of the two or three outstanding figures in ophthalmology and otology in this country during the last thirty years of the nineteenth century. Dr. Roosa was born in Bethel, New York, September 4, 1838, and graduated in medicine from New York University in 1860, serving as house surgeon in the New York Hospital 1861-62. He was for two years an assistant surgeon during the Civil War, later spending a year in postgraduate work in Vienna. In 1863 he became assistant surgeon at the New York Eye and Ear Infirmary. He was surgeon to the Brooklyn Eye, Ear Hospital and to the Manhattan Eye and Ear Hospital. He was president of the International Otological Congress, which met in New York in 1876, and president of the American Otological Society in 1874 to 1876. He was for a time professor of diseases of the eye and ear in New York University. In 1882, as a result of his deep interest in postgraduate education, he founded the New York Post Graduate Medical School and Hospital, of which he was the president until the time of his death in 1908, and this undoubtedly is the chief monument to his memory.

In 1864 he translated von Tröltsch's "Diseases of the Ear," and in 1873 published a treatise on diseases of the ear which was long a standard textbook, going through many editions. The book was the first authoritative treatise on the ear published in this country. It was scholarly in its character, embodying the best that had been accomplished in otology down to the time of its writing. The chapter on the history of otology has never been excelled. Roosa was a constant contributor to the proceedings of the Society in its early years.

At the second annual meeting, 1869, Roosa made a report on the progress of otology.

At the third annual meeting, 1870, in connection with Agnew's cases of trephining of the mastoid, he reported two cases in which he had operated upon the mastoid cells with a probe. This communication contained the first complete account of the mastoid operation since the time of Berger.

These two cases of Roosa and the one of Agnew were among the earliest operations upon the mastoid performed in this

country. Due to the want of knowledge of the indications for performing it on the part of the surgeons of the eighteenth century, the operation received a "black eye" for nearly a hundred years. Roosa and Agnew were pioneers in calling attention to and performing the operation in this country. The indications which Roosa gives in his paper for performing the operation showed a forward looking mind and undoubtedly had much to do with focusing attention upon it. After speaking of Wilde's "Incision of the Mastoid," he said that "the mastoid should be perforated after such an incision whenever the opening is found diseased or a small fistula should be enlarged. It should also be perforated when we have good reason to believe there is pus in the middle ear or in the mastoid cells which cannot find exit by the external auditory canal." Still more striking is his dictum in regard to the operation for chronic mastoiditis. "The mastoid should be perforated in the case of suppuration of long standing with frequent and often painful exacerbations." Roosa made numerous other contributions to the proceedings of this Society, including one in which he called attention to the effect of quinin upon the auditory nerve. His most outstanding contribution was upon the diagnosis between diseases of the internal and middle ear, presented first at the International Otological Congress in Basel in 1886 and before this Society in 1887. In both communications he stated his belief that many cases diagnosed as infection of the tympanum should really be classed among the diseases of the cochlea or acoustic nerves," and stressed the value of the tuning fork in differential diagnosis. He concluded his paper as follows: "In my opinion, people who hear conversation relatively better than the watch or acoumeter, who hear better in a quiet place than in a noise, who hear the tuning fork C<sup>2</sup> better and longer through the air than through the bones, suffer from some lesion of the labyrinth or nerve and not from disease of the tympanum, and I believe a general adoption of this view would stop a great deal of local treatment of the nasopharynx and tympanum and greatly simplify and improve our therapeutics." We may take exception, with our increased knowledge, to the crude methods of diagnosis advanced by Roosa, and his papers do not represent that scholarship found in the papers of leaders of our specialty today.

He was, however, an outstanding figure in otology in this country, and this society owes much to his leadership and support.

A fifth member of the little group of members was O. D. Pomeroy, of New York, a colleague of Dr. Roosa. Dr. Pomeroy was for many years a surgeon at the Manhattan Eye, Ear and Throat Hospital. He was a voluminous contributor to the early proceedings of the society, the first volume including no less than five pages (1868-75). He was the originator of the Pomeroy syringe and was the author of a textbook on diseases of the ear. His work was not of the same high order as that of certain others of his contemporaries, but those who knew him will remember him as a kindly gentleman possessed of a quaint sense of humor.

Of the twelve men who were elected at the organization meeting in 1868, Dr. J. Orne Greene of Boston was an outstanding figure. He belonged to the little group of well trained men who, by their earnest labors, were to put otology in America on the sound, scientific basis on which it has ever since remained. He was twenty-seven years old at the time of the meeting, a graduate of Harvard in Arts and Medicine, and thoroughly trained in his specialty by several years of study abroad. He was for many years one of the surgeons to the Massachusetts Eye and Ear Infirmary, as well as clinical professor of otology at Harvard, for nine years secretary of this Society and its president from 1881 to 1883. During the early years of the society he was a frequent and valuable contributor to its proceedings. The first two volumes of the Transactions contain no less than sixteen communications from him, including papers on "Objective and Subjective Systolic Murmurs in the Ear," "Phlebitis of the Lateral Sinus and Emissary Vein," "Removal of Foreign Bodies by Displacement Forward of the Auricle and Cartilaginous Meatus." Two papers by him should receive particular mention. One was on "The Method of Performing Tenotomy of the Tensor Tympani Muscles," read at the meeting in 1873, and the other on "Neuralgia in and About the Ear," read at the 1874 meeting. The first showed decided original work and the latter paper was a complete and authoritative presentation of the subject, full of merit, even at the present time.



He retired from active practice in 1898, so he will be remembered only by a few of our older members. It is the testimony of those who did know him that he was professionally a careful, painstaking and skillful surgeon, giving his very best thought and care to every case that came before him. As a man he was a modest, kindly and thoughtful gentleman with whom it was a pleasure to be associated.

In 1887 he translated Schwartze's "Pathology and Anatomy of the Ear." Of this, Sexton stated, at the 1878 meeting of the Society, "the work of translating this could not have fallen into more competent hands. He had particularly fitted himself in aural surgery, his contributions being well known everywhere."

During the first twenty-five years of the Society's history 82 members were elected. Many of these deserve mention, but time permits to speak of only five—Albert H. Buck of New York, elected 1870; C. J. Blake of Boston, elected 1869; Herman Knapp of New York, elected 1869; Charles H. Burnett of Philadelphia, elected 1872, and Samuel Sexton of New York, elected 1871. These five men, in addition to the others who have already been mentioned, contributed more to the proceedings of the Society in its early years than any others.

The first of these, Albert H. Buck, was exceedingly modest and retiring, but those who were fortunate enough to know him found in him one who possessed a high degree of scholarship, together with a charming personality. He was born in New York City on October 20, 1842, the son of Dr. Guerdon Buck, one of America's most noted surgeons, and died November 16, 1922, at the age of 81. He was a graduate of Yale, of the class of '64, and of the College of Physicians and Surgeons, Columbia University, '67. Like the others who were the leaders in the infant specialty, he came to his task thoroughly equipped after having served as resident in the New York Hospital and studying for a considerable time with Politzer in Vienna. He soon became an aural surgeon at the New York Eye and Ear Infirmary, which position he retained for many years. He was a careful observer and extensive writer, not merely in his own specialty, in which he brought out a textbook which went through several editions, but also in

other branches of medicine. With his election to this Society he became an active supporter, and during the first twenty years of its existence made no less than seventeen contributions. Among these was one paper in which he recommended the use of applications to the eustachian tube by means of the bougie (page 64, volume 2), and another which showed a careful study of the anatomy of the elephant's ear. He shared with Agnew and Roosa the credit of drawing attention to the importance of surgical interference in mastoid disease, contributing in 1873 a masterly article to the Archives of Ophthalmology on "Diseases of the Mastoid Process—Their Diagnosis and Pathology and Treatment." The pathology there given and the character of the operation which he described was surprisingly thorough for the time. He was, as is generally known, the inventor of the Buck drill and always maintained that he saw no advantage in the use of the chisel and gouge over it.

The American Otological Society has had no more distinguished a representative than the man of whom we next desire to speak. Dr. Clarence J. Blake of Boston, from the time that he became a member of the Society (1869) down almost to the time of his death (1919), but especially in its earlier years, was intimately identified with the proceedings. He combined the rare qualities of being both a student of pure science and also an active clinician, and our Transactions contain many articles by him having to do with both departments of the specialty. With Dr. Blake's cessation from active participation in the work of the Society, the pursuit of scientific otology lapsed until the last few years, when, to the great benefit of the specialty, it has been resumed. Dr. Blake was born in Boston in 1843 and had the same thorough training that those we have already mentioned received. He graduated from the Boston Latin School and Harvard College, and, in 1865, from the Harvard Medical School. Like Greene and Buck, he studied abroad, being a student and assistant of Politzer for a long time. Returning to America in 1869, the year of his election to this Society, he soon became aural surgeon to the Massachusetts Eye and Ear Infirmary and later professor of otology at Harvard. He edited the four volumes of the American Journal of Otology, 1879 to 1882, and was one of the

organizers of the International Congress of Otology in 1876. Our fellow member, Dr. Morse, says of him that "his intelligence was very keen and his mind very active." He occupied a unique position in his profession in Boston and, like that of Dr. Knapp in New York, of whom we shall come to speak later, his name was almost a household word. He was the president of this Society in 1876 and contributed extensively to its proceedings. The list of his contributions took a wide range, as he was interested in every phase of Otology.

The first four volumes of the Transactions represent no less than twenty-four contributions from Dr. Blake. His intimate acquaintance with foreign literature made him peculiarly qualified to prepare the report on the Progress of Otology, which was a feature of the early meetings of the Society. Two of his papers were a summary of the results of original experiments. The first of these, which was presented at the 1872 meeting, was on "The Perception of High Musical Tones"; at the 1873 meeting, "The Diagnostic Value of High Musical Tones." These represent examinations made with König's rods and show the thoroughness and care which characterized all of Blake's work. The conclusions which he arrived at have remained undisputed down to the present day. Pure science, as applied to the ear, was always a subject of great interest with Blake and engaged much of his attention. It is probably not generally known today that he had a conspicuous part in the perfection of the telephone. Vermyme said of Blake, in 1878 (Transactions, Volume 2): "A suggestion by Blake in regard to the means whereby an electric current could be thrown into complex waves by communicating complex vibrations from a membrane like the membrana tympani to the armature of the magnet, gave a clew to the invention of an instrument which has already gained great reputation and has been put to many practical uses. We mean the electric telephone of Graham Bell." This, however, was generally recognized at the time of the invention of the instrument. In the same volume of the Transactions, Vermyme in his report on the Progress of Otology says: "The members of the Society are familiar with the experiments conducted by Blake and Bell while investigating the manner in which articulate sounds were produced, in which the human membrana tympani was

used as a phonautograph, tracing itself on smoked glass, the curves produced by different vibrations." In 1875 he contributed a paper to the Boston Medical and Surgical Journal on the use of the membrana tympani as a phonautograph, in which he described the exceedingly delicate method used in preparing the membrana tympani for registering tracings of sonorous vibrations. The same year, Blake delivered a lecture in London on sound and the telephone, and two years later, 1878, in an article in the American Journal of Otology, page 181, says the use of the membrana tympani was first suggested by him in 1874 for the electric transmission of articulate speech. He gave there an account of the exhaustive work done by him in connection with Bell in perfecting the telephone. While he was interested in acoustics, he was no less interested in the clinical side of otology, and in 1891 read a classical paper before the Society, on middle ear operations, in which he reviewed, in his usual scholarly way, the entire subject leading up to the consideration of the removal of the stapes for the relief of deafness. The operation was first described by Dr. Jack at the same meeting, but the credit of the idea for it he freely gave to Blake. Blake's investigating mind was active down to the time of his death. He was one of the earliest to perform and recommend the mastoid operation, and may be called the father of the blood clot dressing, being the first to bring the subject forward in a contribution read at the thirty-first annual meeting of the Society in 1898.

There was elected the same year as Blake, 1869, a man in many respects very unlike him and yet resembling him in his indefatigable ability for work and in what he accomplished for otology in this country. Indeed it can be said that Dr. Herman Knapp is entitled to be regarded equally with Dr. Blake and Dr. Roosa as the most outstanding representative of our specialty in America. Born in Danbold, Prussia, March 17, 1832, he was already thirty-seven years old when he came to America, and in the forty-two years that he lived here (he died April 30, 1911) he accomplished more than a half dozen men of ordinary capacity. He was thoroughly trained for his work, being a graduate in medicine from Giessen in 1854, afterward visiting most of the great medical centers of Europe and studying under Helmholtz. He founded the chair and

became the first professor of ophthalmology in the University of Heidelberg, 1864 to 1868. He was not satisfied, however, with this high honor and resigned to come to America in 1869. On his arrival, he found a wide field for the exercise of his skill in ophthalmology and otology, and founded the same year the New York Ophthalmologic and Aural Institute, of which he was the head for thirty-eight years, during which time over 345,000 patients passed through it. All the important cases were seen personally by him. He was professor of ophthalmology in the College of Physicians and Surgeons, 1888 to 1892. His practice was an enormous one, and his help was sought from all parts of the continent. Perhaps his most conspicuous contribution to medicine was the Archives of Ophthalmology and Otology, which he founded in 1869 and which was published later in two separate journals, one devoted to ophthalmology and one to otology. The latter appeared simultaneously with the *Zeitschrift für Ohrenheilkunde* in Germany. Knapp was a regular and conspicuous attendant at the early meetings of this society and contributed extensively to its proceedings, either by papers or in discussion. One of the earliest successful reports of operations upon the mastoid was made by Knapp at the 1879 meeting when he described a case of recovery in one month following the use of the Buck drill with rubber dressing, under the heading "Trephining the Mastoid in a Case of Acute Suppurative Otitis Media, Entire and Rapid Recovery" (Volume 3, page 208). Three years later he contributed another important paper on "Indications for Opening the Mastoid Process Based on Some Recent Observations" (Volume 3, page 208, 1884). This shows a rapid development in the surgery for the relief of mastoiditis. In this Knapp refers to the skepticism exhibited by the majority of members present of the value of operation. By this time he had come to the conclusion that the operation was indicated in certain cases when the drum membrane was intact. The method of operation he described is not unlike the operation of today, the chisel being the instrument which was preferred by him rather than the drill. At the meeting in 1887 he presented an exhaustive report, as chairman of a special committee with Dr. Prout and Dr. Roosa, on the "Examination of the Power of Hearing and How to Record Its Results,"

while at the 1880 meeting he contributed an excellent paper on "Some Remarks and Observations on Bone Conduction," based on his own observations and showing the thorough training he received from Helmholtz. Knapp was of medium height, he had bright, sharp, blue eyes, a short, sandy beard. While he was a thorough master of English, his German birth always gave to his speech a slight, not uninviting accent. He was a man of few words and at first acquaintance gave the impression of gruffness. Those who knew him best know that this was not true and that he was in his treatment of the poor most democratic. He occupied a unique position in his specialty, and the high ideals that he had for it did much to make it what it is today.

Only second to what we may style the triumvirate just mentioned, Roosa, Blake and Knapp, stands in number and importance of his contributions to the early proceedings of the Society, Dr. Charles H. Burnett. Dr. Burnett was another example of one of those who had thoroughly prepared himself for his life's work, a striking contrast to those of the present day who are demanding admission to the specialty after a three months' course in some postgraduate college. He was born in Philadelphia in 1842. After graduating from Yale in 1864 and in medicine from the University of Pennsylvania in 1867, he served as intern in the Episcopal Hospital. He then spent two years abroad in the study of otology, working with Helmholtz and Arnold. He began the practice of his specialty in Philadelphia in 1871, where he lived until the time of his death in 1902. His first contribution to otology appeared in 1871 in a paper giving his investigations concerning the mechanism of hearing and the membrane of the "round window," followed the next year, 1872, by a paper on the investigations of the membrana tympani of the mammalia. Both of these papers showed careful preparation and distinct merit. He became a member of the Society in 1872 and was a frequent contributor to its Transactions. During the first twenty years of its existence he made no less than twenty contributions to its proceedings, writing two carefully prepared reports on the Progress of Otology for the years 1873 and 1874. A paper read at a special meeting in 1888 on "Chronic Purulent Inflammation of the Tympanum" reveals by the absence of all

pathology and by the slight reference to surgical interference, the dense ignorance of the profession on this subject only a little over a generation ago. He published a treatise on diseases of the ear in 1877 which was regarded by Dr. Roosa, ten years latter, as "among the most valuable textbooks on the ear of the present day." This was followed in 1893 by his "System of Diseases of the Ear, Nose and Throat." His acquaintance with current otologic literature was most intimate. Of him Randall says in his biographic sketch: "It was rather his characteristic to be an aggressive advocate of some of the newer views in otology, and he never hesitated to reverse his earlier judgments, when he or others proved them untenable, and to criticise vigorously whatever he disapproved. Of small physique and giving the impression of poor health, his trenchant criticism in discussion at times suggested acerbity of disposition, but all who knew him intimately bear witness to his punctilious courtesy and rectitude of purpose. He has been a real force in shaping otologic knowledge and practice in America, and the profession in general as well as his colleagues in this Society has sustained a loss in his death."

There were many others who took an active part in the early meetings of the Society. Time permits us, however, to speak of only one other, Dr. Samuel Sexton of New York. Dr. Sexton was born in Ohio in 1833 and died in New York City in 1895. He graduated from the University of Louisville in 1856. Serving in the Army during the Civil War, he came to New York and engaged in the practice of Otology about 1862. Unlike most of the others of whom we have spoken, he did not study abroad but possessed all of the student qualities in his nature and studied his specialty particularly from the standpoint of the general practitioner. In 1882 he published a paper on the cause of deafness among public school children which was widely circulated and generally quoted. In 1887 another contribution, "Injury to the Ear Caused by the Blast of a Bursting Shell," appeared. He became a member of the Society in 1871 and was a constant attendant at its meetings. The first four volumes contained nineteen contributions from his pen. Many of these are of decided value. The most important, however, were the papers on the treatment of chronic suppuration of the middle ear by means of removal

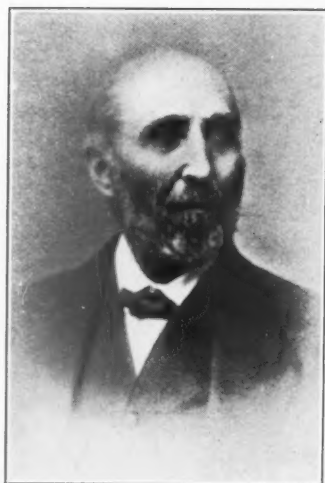
of the ossicles. His first paper on this subject was read before the Society in 1886. In 1888 he elaborated the method with a paper on "Excision of the Drumhead and Ossicles for the Cure of Chronic Purulent Inflammation of the Attic of the Tympanum." These papers did much to give the operation the popularity which it possessed for a number of years. Sexton was a hard worker and a close observer. In his acquaintance with his professional brethren he was the soul of honor.

Such is the story, briefly told, of the beginnings of this Society. The later decades, from 1890 to the present time, are known to us all. The record is no less creditable. Many who contributed to make it are of us and still with us. Neither time nor place permit to dwell on what they have done to promote otology in America. Their works speak for themselves.

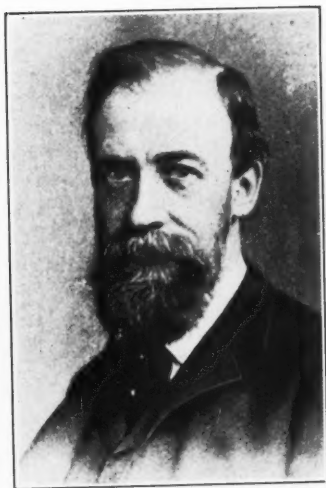
To rest content with what has been accomplished is to go backward. Unknown realms are yet to be discovered as well in otology as in the Polar regions. We shall hear at this meeting of ways and means which make the prospects of doing this very bright.

The future of this Society and of otology in America is full of encouragement. This meeting promises to mark the beginning of a new era in the study and solution of many of the intricate problems of this specialty. It is our confident hope and expectation that with our present methods of investigation and with the means to be provided for carrying them on, the next fifty years will witness, under the lead of this Society, marvelous and undreamed of achievements in the specialty of otology.

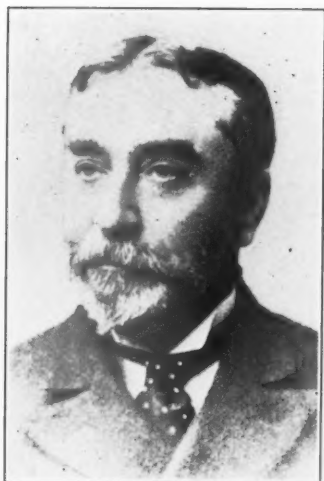




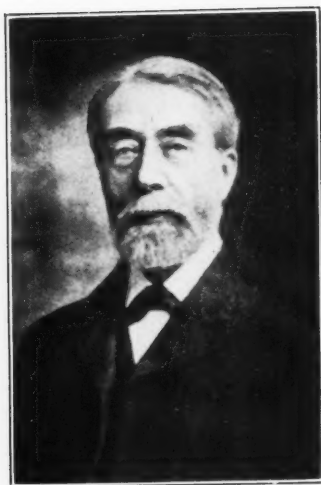
E. WILLIAMS, M. D.



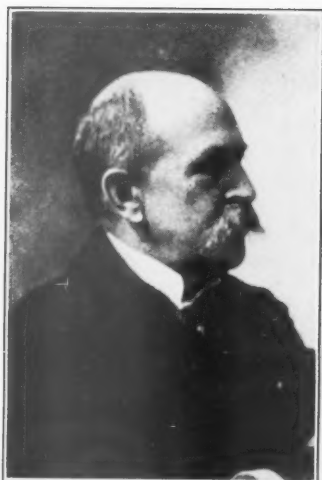
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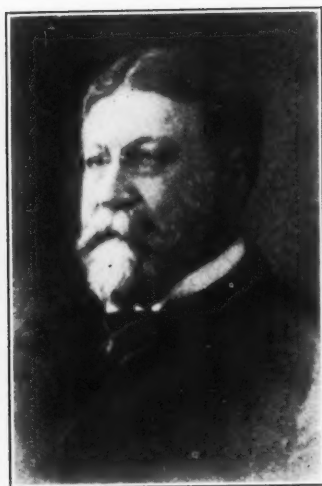
DANIEL B. ST. JOHN ROOSA, M. D., LL. D.



HERMAN KNAPP, M. D.



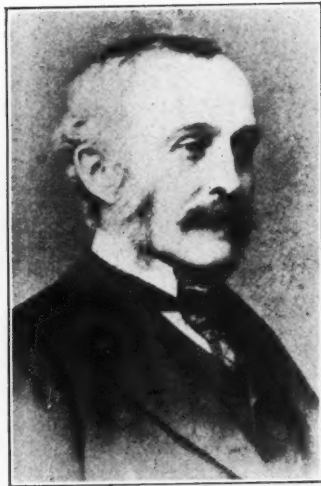
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XXIV.

THE REPORT OF A CASE OF HEREDITARY SYPHILITIC DEAFNESS COMPLICATED WITH BILATERAL MIDDLE EAR SUPPURATION AND MASTOIDITIS.\*

By GEORGE W. MACKENZIE, M. D.,

PHILADELPHIA.

The reason for reporting this case is because of its unusual character. Uncomplicated hereditary syphilitic deafness is not at all uncommon, neither is bilateral middle ear suppuration with mastoiditis, but the combination of the two conditions appears to be rather infrequent, since this, so far as I am aware, is the first case of its kind to be reported. On the other hand, it would seem that those individuals who are afflicted with hereditary syphilis ought to be more susceptible to all sorts of ailments, including middle ear suppuration, than others who are fortunate enough to be free of the syphilitic taint.

The case, J. B. W., age 34 years, occupation, painter, was first seen May 17, 1919. The family history could not be obtained, since the patient was an orphan from early childhood. He appeared to be of muscular build and at one time had been in the prize ring. He was full of pep and cheerful in spite of his misfortune.

Anamnesis: The patient claims to have had a double mastoid operation performed at Scranton, Pa., in 1910, and attributes his present trouble to these operations. Dr. J. J. Sullivan, Jr., of Scranton, was written to for further information. His reply reads as follows:

"Weaver case: Ten years (10 yrs.) ago a patient came to me suffering from an acute mastoiditis, bilateral, suppurative. Both ears were absolutely deaf. Pain and tenderness over both mastoids, profuse discharge from both ears. He was so deaf he looked stupid. I do not remember whether he gave previous history of deafness before operation or not, but subsequent

\*Read before the Buffalo Laryngological Society, February 2, 1926.

evidence leads me to believe he had been deaf before operation. The operation was a simple complete mastoid. Large amount of free pus and débris found in both mastoids. Patient made an uneventful recovery. Wound healed nicely, but did not recover his hearing."

The patient claims that prior to 1910 he had never had any trouble with his ears; but since then his hearing "is constantly getting worse." He states that his trouble first started with an abscess in the throat. For the last two years his left ear has been running continuously. The right one is dry. He further states that he had adenoids "broken down" and an operation performed on his nose. He thinks it was a "submucous." He says that he is not subject to colds. His principal complaint is a feeling of unsteadiness on the scaffold when painting and he cannot walk straight in the dark.

While sitting in front of the patient eliciting the history, the writer was struck with his color, that dirty gray pallor so often seen in syphilis. Next, with reflected light from a head mirror, a definite haziness of both corneæ, indicative of interstitial keratitis, was observed. These findings naturally suggested an examination of the teeth, which resulted in the finding of notched incisors, and typically peg shaped cuspids and bicuspid.

The functional hearing tests revealed the following findings: Weber not lateralized. The duration of bone conduction taken at the midline of the forehead was definitely shortened. The Schwabach (bone conduction taken on the mastoid), in this case over the antrum region, showed a fair amount of shortening, 20 seconds on the right side and 22 seconds on the left, with a fork that is normally heard on the mastoid for 70 seconds. The Rinne was positive on both sides, but considerably less positive than normal. The air conduction with the same fork was shortened 60 seconds on the right side and 55 seconds on the left. The hearing for the low C (64 d. v.) fork was quite short on both sides; furthermore, the hearing for the high  $c_4$  (2048 d. v.) was very short on both sides. The speaking tube and noise producer test was next tried, when it was found that with the tube in the right ear and noise producer in the left, the patient was able to repeat conversationally spoken words 100 per cent correctly. With the speaking tube

in the left ear and noise producer in the right, he was able to repeat conversationally spoken words 50 per cent correctly.

Otoscopic examination: Right ear, membrane is intact, brilliant and slightly opaque. It is possible to see the long process of the anvil through the drum membrane, but less distinctly than normal. There is no retraction and the membrane moves normally with the Siegel instrument. By Politzer inflation the membrane moves freely outward and goes back to its primary position promptly, showing the eustachian tube to be patulous and the membrane resilient.

Left ear: The upper wall of the canal, near the membrane, shows a few small scattered white spots, which proved to be nothing of importance after cleansing the canal. The upper half of the drumhead is intact. The hammer handle is visible and is in normal position. There is a slight amount of purulent nonodorous secretion presenting from a small perforation just below the umbo region.

The patient was next sent to the Philadelphia Clinical Laboratory for a Wassermann blood test. The report came back on May 20th, "negative to three antigens."

He next reported on the 24th, when the examination was continued. Examination for spontaneous nystagmus revealed a definite nystagmus of the mixed type, to the right side with the patient looking straight ahead, 1 mm. length of excursion at the equator, repeating itself every two seconds.

After-turning nystagmus test findings: After ten turns to the left, with the head erect, there was no change in the character or intensity of the nystagmus from that which was present before turning, nor was the patient made dizzy. In other words, the reaction was negative. After ten turns to the right, with the head erect, there was no change in the character or intensity of the nystagmus from that which was present before turning, nor did the patient experience any dizziness. The reaction was therefore negative. The turning tests were repeated with increased number of turns up to twenty, first to one side and then the other, with equally negative results.

After-turning tests were next made with the patient's head inclined forward, turning first to one side and then the other. Again there was no reaction to be noted on either side, nor was

vertigo produced by the turning, no matter how long he was turned.

The caloric test was negative. Syringing each ear separately with cold water as low as 60 degrees for an indefinite time and with the head in different positions produced no change in the character (plane of eye movements, direction or intensity) of the nystagmus which was present before making the tests, nor was any dizziness produced.

Compression and aspiration nystagmus was next tried. Strong, steady compression of air in the external canal of the right side, making use of the Gellé outfit, resulted in no change in the existing nystagmus, nor did it produce any vertigo. Neither did aspiration.

Galvanic reaction: Right ear—with the kathode, 3 M. A. of current produced a definite increase in the existing nystagmus to the right side. With the anode, 5 M. A. of current reversed the direction of the nystagmus—i. e., produced a nystagmus to the left side.

Left ear—with the kathode, 5 M. A. of current produced a nystagmus to the left side. With the anode, 3 M. A. of current produced a definite increase of the existing nystagmus to the right side.

Patellar reflexes on both sides were prompt and of normal excursion; Rhomberg doubtful. The gait, forward and backward with open eyes, is slightly uncertain and broader than normal; with closed eyes, the gait is very uncertain and broad. The patient is quite aware of this uncertainty in his gait and is embarrassed by it, fearing that people might think he had been drinking.

He next reported on June 2. Between this and the last visit he had been confined to the house with a sore throat. The examination was continued on this visit, checking up on the findings previously made. The corneal opacities were studied more closely with the ophthalmoscope and dilated pupils, when the diagnosis of bilateral interstitial keratitis was confirmed.

June 16. Removed remnants of adenoids under local anesthesia.

June 26. Patient claims he feels better since the operation. He has been receiving treatment with mercurial inunctions,

after Professor Finger's method, for thirty rounds, followed by potassium iodid in increasing doses. Under antiluetic treatment his general appearance improved rapidly, including his color; furthermore he had added a few pounds to his weight. Unfortunately the blood examination further than the Wassermann test was not made.

September 5. The patient reports that he "never felt better in his life," and he had gained ten pounds in weight. The head noises are bothering him less. He claims to be able to hear newsboys 40 feet away. Notwithstanding this favorable report, the functional tests reveal but little, if any, improvement. The speaking tube and noise producer test shows a slight gain in hearing on the left side, for now he is able to repeat conversationally spoken words 100 per cent correctly.

October 17. The patient reports that he feels well, the left ear has stopped discharging and the noises in the head have ceased. He claims to be sleeping better than formerly and has a much better appetite. At this time he was given mercuric iodid, one-tenth of a grain three times daily after meals. The functional hearing tests did not show the amount of improvement that one would expect to find, judging from the patient's report. However, he is able to recognize conversationally spoken words through the speaking tube 100 per cent in each ear; furthermore, he is able to recognize some words spoken in a whisper through the tube. There is no change in his vestibular reactions from that found earlier. It is well to mention the fact that the patient's hearing ability varies appreciably on different days, roughly estimated at 20 per cent.

He was last seen on January 10, 1920, when he reported that his hearing was "very punk," that spoken words seemed to sound "jumbled." He appeared to be very much discouraged and has not reported since.

#### COMMENTS.

I. That this patient was afflicted with labyrinthine deafness is supported by the following facts: Impairment of both air and bone conduction, together with a positive Rinne on both sides, and considerable shortening of perception for the high as well as the low tones.

II. That the pathologic process in the labyrinth was a diffuse and not a circumscribed one is evidenced by the negative findings from the turning and caloric tests. In fact, the loss of function in the semicircular canal apparatus was even more complete than the loss of function in the cochlea.

III. That the pathologic process was not only a bilateral diffuse labyrinthine affection but did not extend inward beyond the labyrinth is evidenced by the fact that the vestibular nerve reacted to the anodal and the cathodal current on both sides comparable with that found in individuals possessing normal labyrinths.

IV. That the labyrinthine affection in this case was labyrinthitis syphilitica tarda of hereditary syphilitic origin is evident from the fact that the local findings were characteristic for this disease; besides, the patient gave other evidences of hereditary syphilis, notably the corneal opacities, characteristic of interstitial keratitis and the so-called Hutchinson's teeth.

V. That a part of the impairment of hearing was possibly due to pathologic changes in the middle ear is suggested by the history of middle ear abscesses with mastoiditis and the aggravation of his hearing trouble, following a so-called cold, on one or two occasions. The Schwabach and Rinne tests are less dependable in this case, because the fork could not be applied satisfactorily on account of the absence of the major portion of the mastoid following the mastoid operations performed in 1910.

VI. That the negative blood Wassermann in this case does not contradict the presence of syphilis is evident from the fact that it is found just as often negative as positive in the hereditary form. Browning and Cruickshank (*Glasgow Medical Journal*, LXXXIX, 81, 172) found, in a large series of cases of congenital deafness from syphilis, that the reaction was negative in fully 50 per cent.

VII. That the deafness was progressive in spite of treatment merely shows that the disease was behaving "true to form" in this case as it does in the majority of this kind.

VIII. That the operations on the mastoids in this case had nothing to do with the patient's deafness is self-evident, since the hearing defect was due to a different and explainable lesion

in the inner ear—labyrinthitis syphilitica tarda; while the element of obstructive deafness was almost negligible. It is possible it might have been more pronounced had the mastoids not been operated on.

Although the combination found in this case is a comparatively rare one, in the aggregate there are probably quite a few others just like it.

## THE NATURE OF NYSTAGMUS.\*

BY EUGENE R. LEWIS, M. D.,

LOS ANGELES.

The term "nystagmus" is frequently used to denote any kind of jerking or unusual movements of the eyes, including spasmodic tremors, searching and irregular movements of many kinds. This discussion does not include consideration of so-called "miners' nystagmus," or of any of the abnormal eye movements observed in association with peripheral lesions, with low visual acuity, with epilepsy, with multiple sclerosis and with certain neuroses. Only the natural types of eye movements will be considered in this exposition of certain physiologic motor and sensory relations.

Ordinary eye movements under practically all conditions consist of two main phases, the fixation of gaze upon some object and the quick movement of directing the gaze to the next object of fixation. If the objects fixed upon are still, the eyes are still during the first phase; if the objects are moving, the eyes move with the moving objects. The movement of directing the gaze upon the next chosen object of fixation is always a quick jerk.

Bright objects, brilliant colors, or lights, against less striking background, attract the fixation of the gaze, either consciously or subconsciously. If a cylinder of solid color be rotated before the eyes, no special eye movements will result; but if the cylinder be striped in striking colors, the eyes will execute a series of movements as long as the rotation continues, and the movements will be specifically (1) a slow pull in the direction of the rotating stripes, (2) a quick return jerk in the opposite direction. This is normal physiologic nystagmus—the result of fixing the gaze upon a stripe attracting visual attention as long as it is in sight, and successively fixing the gaze upon other stripes as they rotate into and out of view. During a

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railroad journey, gazing out of the window causes the same series of eye movements, the gaze being consciously or subconsciously fixed upon landscape objects as they come into view successively. This is called "railroad nystagmus"; its slow pull is always in the direction of the passing objects, the quick jerk, in the direction of the train's progression; the plane of the eye movements is always that of the train movement responsible for it.

While nystagmus is an ocular phenomenon, its nature is such as to be understood only when studied from an approach based upon broad general physiologic considerations. The "pattern" concept put forth by Dr. Samuel D. Ingham differs radically from those previously advanced in dissertations upon the subject of nystagmus; it takes no cognizance of the relations conceived to exist between certain ampullar end organs and certain extraocular muscles, but recognizes much broader and more fundamental biologic principles and physiologic facts.

The evolution of species attests the truth of certain biologic principles, among the first of which is that natural adaptations tend toward higher efficiencies in inherited characteristics. Complicated motor coordinations, varied by individually acquired modifications in generation after generation, come gradually to be laid down in the germ plasm as more or less definitely predetermined "reaction patterns." This conception of "reaction patterns" constitutes a fundamental explanation of practically all instinctive acts and generic automatisms. Once the "pattern" comes into circuit the neuromuscular apparatus performs in more or less exact accordance with the pattern requirements. The patterns represent coordinations acquired through age-long lineage adaptations to environment requirements. The swimming movements of baby fish, the flying movements of baby birds or the sucking movements of newborn mammals are examples of spontaneous motor performances fundamentally similar, regardless of familial relations. In this simple biophysical concept of inherited tendencies toward the maturing of certain cell groups according to definite generic or species "patterns" is found an explanation of the similarity of inherited motor reactions. Just as the "anlage" of the gonads are predestined to attain maturity only at the age of puberty, so are the "anlage" of other tissues predes-

trained to attain definite functional stages at various development periods. In the case of the sucking apparatus of mammalian infants, the actual cellular neuromuscular circuits are adequately developed at birth; in the case of the flying mechanism of baby birds, the actual cellular neuromuscular circuits are not completely developed until the fourth or fifth week after hatching. In both instances, further developmental maturity brings with it additional technical facility and physical capabilities, constituting acquisitions on the part of the individual. As a result of such acquisition, the four-weeks-old baby is able to nurse better and longer than it could at birth; the ten-weeks-old fledgling is able to fly more accurately and longer than it could at its first flight. But the "pattern" of each of these coordinations was laid down at the moment of beginning of embryonic existence of the individual, with a certain definite predestination of its attaining workability with the completion of the circuit at a certain stage in the development of the individual. With the higher and higher development of visual efficiency of each of the two eyes comes the necessity of binocular fixation of the gaze upon the same object at the same moment, in order to avoid the confusion of simultaneous distinct, but dissimilar, visual impressions. A marked degree of conjugation of eye movements exists at birth by virtue of an inherited coordination pattern; but the individual acquisition of higher and higher degrees of conjugate ocular coordinations proceeds hand in hand with other mental developments until, during the period of infancy, fully established binocular fusion is evolved. Eye movements actuated by the requirements of visual efficiency demand certain anticipatory direction of gaze of each eye simultaneously, prior to, or in process of, fixation of gaze of sufficient duration to permit the cerebral registration of a clear visual image. During sustained movement of an individual in one direction, this results in an arrest of the gaze as it fixes upon a certain object, which is thereupon clearly sensed visually before falling behind out of view as the body progresses; followed by a quick jerk of the eyeballs forward in the direction of the body movement, in fixing the gaze afresh upon some newly chosen object; and so on during the continuation of the sustained forward movement of the individual. This succession of

slow reverse pullings of the eyeballs and of quick return jerks resulting from such sustained forward movement of the body is physiologic or "railroad nystagmus." This type of "pattern" has developed, obviously, in answer to the demands of visual efficiency for the body in motion. Rotation to the right causes "railroad nystagmus," whose quick jerk is to the right; its plane is always that of the body movement responsible for it.

With eyes closed, or with unseeing eyes, a normal individual on being subjected to the same sustained movement develops certain involuntary or reflex eye movements which are not actuated by vision, but depend upon nerve impulses originating independently in the motion sensing apparatus of the ears. These eye movements consist of a slow pulling followed by a quick jerk in the opposite direction; they continue as long as the impulses continue to emanate from the vestibular end organs; they occur in a certain definite direction and plane, the direction of the quick jerk always in that of the rotation, and the plane of the eye movements always in that of the body movement responsible for it. This nystagmus is called "vestibular nystagmus during rotation."

Thus two special senses, each functioning individually, each concerned with adjustments relating to movement, each having its special reaction to every form of movement, are seen to be so intimately related as to have a "common reaction pattern," which acts as an interlocking director. In other words, a certain motion will produce visually in the absence of vestibular function, or vestibularly in the absence of visual function, the identical series of eye movements called nystagmus.

Certain commentators have expressed doubt as to the vestibular apparatus subserving a special sense function, despite the functional characteristics which seem to fulfill the requirements of a special sense; the identity of the vestibular eye movement reflex with physiologic visual nystagmus constitutes a pertinent additional indication of the special sense nature of this motion sensing apparatus.

This "vestibular nystagmus during rotation" is identical with the "railroad nystagmus" caused by this rotation. The physical stimulation of the end organ endures only until the fluid within the canals has taken up the rotation movement; as the vestibular nystagmus during rotation only continues as long

as the impulses continue to emanate from the end organs, it is apparent that with continued rotation at a constant speed this nystagmus gradually ceases. On ceasing to rotate, with eyes closed or unseeing eyes, the normal individual develops certain involuntary or reflex eye movements which are not actuated with respect to visual considerations but depend upon nerve impulses originating in the vestibular portions of the ears. These eye movements consist of a slow pull, followed by a quick jerk in the opposite direction; they continue as long as impulses continue to emanate from the vestibular end organs; they occur in a certain definite direction and plane, namely, the slow pull always in the direction of the rotation, the plane always in the plane of the head coinciding with the plane of the rotation. This nystagmus is called "vestibular nystagmus after rotation"; it is, in direction, the reverse of the railroad nystagmus occurring during the rotation responsible for it.

Resumé.—Nystagmus is a physiologic motor phenomenon. This phenomenon may be normally elicited by ocular stimulation or by aural stimulation, or by both.

In either event the resulting eye movements consist of two phases: a slow pull in one direction, followed by a quick jerk in the opposite direction. The direction and plane of these eye movements are determined in accordance with certain definite coordination "patterns."

Movements to which the head is subjected call into circuit the same "pattern," with the result that eye reaction movements, whether from visual or aural initiation, are identical in direction, in plane and in phase.

"Railroad nystagmus" is identical with "vestibular nystagmus during rotation." This identity seems to constitute a very pertinent indication that the vestibular mechanism comprises the elements of a special sense apparatus no less worthy of the name "special sense" than that of the ocular mechanism.

730 So. CATALINA ST.

XXVI.

ACTINOMYCOSIS OF THE TONGUE WITH CLINICAL PRESENTATION OF A CASE.\*

BY JOHN MONRO BANISTER, A. B., M. D.,

OMAHA.

Primary tongue infections by the ray fungus in the human subject are of sufficient rarity to justify the reporting of each individual case encountered.

One hundred and twenty-seven patients infected with actinomyces were examined at the Mayo Clinic from January 1, 1910, to January 1, 1921. In only three instances did it occur primarily in the tongue, according to New and Figi.<sup>1</sup> These observers state that in reviewing the literature they were able to find only thirty-eight cases of primary infection of the tongue, counting their own three cases in this list. They have since found three other cases. In discussing this subject of actinomycosis of the tongue, the writer desires in advance to express his indebtedness to papers by New and Figi<sup>1</sup> of the Mayo Clinic, by Denis Bonnet<sup>2</sup> of Lyons, France, and by R. A. Barlow,<sup>3</sup> to which papers the reader is referred.

Bonnet believes that the rarity of primary tongue infections is due to two causes, an anatomic and a histologic cause. To quote his words from his article in *International Clinics*, before indicated: "The tongue is protected by the double barrier formed by the lips, and especially the gums and teeth, and consequently is relatively well protected from those foreign bodies which are usually the carriers of actinomycosis. On the other hand, it is admitted that the connective tissue is the preferred soil for the evolution of the germ, and in the tongue this tissue is present in very small amounts. The organ, protected by a thick mucosa and formed by a mass of tightly woven muscular fibers, which are directly united with the mucosa without any

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intervening submucosa, offers a very unfavorable soil for the development of the parasite."

The infection is considered to be transmitted to the human subject, as a rule, through vegetable matter harboring the actinomyces, and hence is chiefly met with in farmers, stable hands and tenders of cattle, whose occupation brings them in close contact with vegetables and infected animals (Barlow).

According to Bonnet, "In the immense majority of cases the subject has been chewing a spear of wheat, oat or barley which excoriates the tongue and inoculates it."

The primary lingual lesion is an indurated and tender nodule, most generally located in the anterior portion of the tongue. The mucous membrane covering the nodule is tense. The mass increases in size at the expense of the muscles, which atrophy. (Barlow.) Lymphatic enlargement is absent, as a rule. There may be abscess formation due to secondary invasion of staphylococci. The presence of the sulphur bodies and mycelial fibers characteristic of the ray fungus will definitely signify actinomycosis.

In making a diagnosis the habit of chewing grain beads or spears of grain or other vegetable matter, the habitual care of animals, a chronic course manifested by the disease, and the absence of general lymphatic involvement, are matters of importance. (Barlow.) Recognition of the sulphur bodies and ray fungus under the microscope will definitely establish the diagnosis.

It should be noted, however, that it is not always possible to find the sulphur bodies,<sup>4</sup> but in such cases the presence of the mycelial fibers will be sufficient, with other clinical evidence, to fix the diagnosis. The early symptoms are not especially severe. There may be some feeling of soreness and thickness of the tongue, which has given rise to the term "wooden tongue" in this connection. Pain is rarely present as a definite symptom. The nodule increases in size slowly, though at times the tissues surrounding the nodule, owing to the irritation produced by the same, may swell quite rapidly, causing decided inconvenience in mastication and deglutition, and after a few days subside, leaving the nodule of its original size.

The methods of treatment most commonly advised are the use of incision and drainage, the internal administration of potassium iodid and the application of radium to the nodule. The application of copper sulphate to the incision and swabbing of the wound with silver nitrate solution are also advised (Barlow).

The results of treatment are satisfactory when measures of relief are instituted before multiple abscesses have occurred and when the nodule is not too deeply seated near the base of the tongue (Barlow).

As regards the mode of action of potassium iodid, D. G. Berard, Bordeaux, France, holds that this drug is not a specific and does not act upon the actinomyces directly, but that it modifies the tissues involved, perhaps by increasing the resistance.<sup>3</sup>

#### CASE REPORT.

Mr. L. J., about eighteen years of age, consulted me on September 25, 1925, on account of a persistent and increasing nodular growth on the left side of the dorsum of the tongue and well back toward the base. This growth first attracted attention two years ago. It has been growing ever since, and at times the surrounding tissues undergo a marked swelling, which, while not painful, seriously interferes with the movements of the tongue. These swellings subside in a short time. During these two years he had been seeking relief of many physicians without avail. All sorts of diagnoses have been given, but from the patient's statements, no one had suggested a fungous origin of the growth. At the time of his first coming to our office, my colleague, Dr. H. B. Lemere, suggested the probability of the lesion being due to a fungus and, acting upon this hint, further investigation was carried on. We both suspected actinomycosis. He was referred to Dr. A. S. Rubnitz for laboratory examination. Dr. Rubnitz, who will present his findings to this meeting today, could not find the characteristic sulphur bodies, but did discover the mycelial fibers at the first examination, and has proved their presence repeatedly since.

The pathologist diagnosed the lesion as being a manifestation of actinomycosis. The young man lives on a farm and has

been thrown into a very constant relationship with cattle, which is suggestive of the source of infection. The patient was at once put upon increasing doses of potassium iodid and for some time has been taking 210 grains a day.

Under this medication the nodule has decreased in size, and the patient has been much more comfortable. I trust that views concerning further treatment will be brought out in the discussion which will be of value, as, for instance, opinions concerning the efficacy of deep incisions, diathermy and applications of radium and the roentgen ray.

The patient is here, and examination of the lesion on the part of those present is invited.

#### BIBLIOGRAPHY.

1. Gordon B. New and Fred A. Figi: American Journal of the Medical Sciences, April, 1922.
2. Denis Bonnet: International Clinics, Vol. III, Thirty-first Series.
3. R. A. Barlow: The Laryngoscope, April, 1925, and Practical Medical Series, 1925.
4. M. G. Wahl: Section on Pathology and Physiology, American Medical Association, 1923.
5. D. G. Berard: International Clinics, Vol. III, Thirty-first Series.

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#### PATHOLOGIC REPORT. BY DR. A. S. RUBNITZ, OMAHA.

I first saw this patient, at the request of Dr. Banister, on September 25, 1925. His tongue was markedly thickened at the left base and more or less adherent to the floor of the mouth on the left side. It was with some difficulty that he could open his mouth wide enough to permit a satisfactory examination. Necrotic pieces of tissue were seen to be plugging some sinuses. When an attempt was made to get one of these pieces out with a platinum loop it was found that they were attached by a pedicle to the mucosa, and those necrotic pieces of tissue were the terminations of enlarged papillæ of the tongue. One such piece was snipped off with scissors and crushed between two slides. It consisted of epithelial cells, leucocytes and numerous filamentous structures, with occasional sporelike bodies. There was no definite arrangement of the filaments, nor was there any clubbing at the periphery. On exploring the sinuses with a platinum loop, they were found to be quite deep (3 to 5



cm.). Small pieces of necrotic material were pulled out from the bottom of the openings and smears were prepared from them. The stained smears contained numerous bundles of mycelia. Some of the mycelia, especially the more slender ones, did not take the Gram stain, while the thicker ones were Gram positive. While the majority of the mycelia were individual ones, yet there were a good many that showed definite branching. A few tubes of glucose broth were inoculated with the necrotic material, but we were unable to reproduce the mycelia in culture.

The patient was seen again on October 10, 1925. At this time the tongue was less swollen, and the patient could open his mouth with greater ease. Some of the sinuses that were not accessible at the first examination could be explored better now. From the bottom of those sinuses little granules of pus were extracted, simulating the so-called actinomycetes granules or "Drusen." Crushed granules in 10 per cent sodium hydroxid solution showed compact filamentous masses but without characteristic radial arrangement; nor were there any club-shaped bodies seen. There were, though, numerous sporelike bodies of conidia present. Stained specimens again consisted of Gram positive and Gram negative filaments, with a good many of the heavy filaments showing active segmentation. At this time a second attempt was made to culture the pus granules. The method recommended by Wright<sup>1</sup> was strictly carried out, and six tubes of glucose agar were inoculated, but outside of contaminating bacteria nothing grew there. Since then examinations were made several times with approximately the same results. Mycelia were obtained at each and every examination.

Our diagnosis of actinomycosis in this case we feel is justified, although we expect some criticism. We want to consider, first, the chronicity of this condition; the lesions had been there for so long that unless we get down surgically to the bottom of those sinuses we cannot very well expect typical, viable organisms. The fact that the lesion is in the oral cavity, where every minute spot is inhabited by numerous bacteria, makes the culturing also difficult. This was our deduction after several unsuccessful attempts in culturing the organisms, and we were gratified in finding a corroborative statement to that

effect by Drs. New and Figi<sup>2</sup>, in an article on "Actinomycosis of the Head and Neck," where they state, "In actinomycosis of the tongue, or if there is a good deal of secondary infection, a diagnosis must depend on detailed study of the tissue removed for microscopic section."

From textbooks one usually gets the impression that actinomyces are cultured comparatively easily, but when you go back to the original works you change your mind, and you are not discouraged if your first attempts fail. Bostroem,<sup>3</sup> who was one of the foremost contributors to the study of actinomycosis, makes the following remarks: "It is of the greatest importance to inoculate as many tubes as possible, for it is impossible to foretell whether material placed in tubes contains living microorganisms or not." In one case he found that although 85 tubes had been inoculated not a single actinomyces colony developed. Some tubes remained completely sterile and others showed only a few contaminating bacteria. Regarding the clubbing of mycelia, our knowledge is also not complete. Most of the biologists consider the clubbing as a protective mechanism against the tissue secretion—i. e., given a case of actinomyces invasion of the tissue, the tissue fluids will attempt to disintegrate the filament, and as a result of that the ends of the filaments will form thickenings, which are more resistant to the destructive action of the tissue fluids. The absence of clubbing, therefore, does not necessarily mean that we are not dealing with actinomycosis, as in a case reported by Wright,<sup>1</sup> where all he found was compact filamentous colonies without the characteristic clubbing at the peripheri. There has been a good deal of discussion among the bacteriologists as to the nomenclature and classification of actinomycosis, some taking the "actinomyces bovis Harz" as the type, while others extend the class to nocardia in general. The following is the description used by the committee of the American Society of Bacteriologists, 1920:<sup>4</sup> "Organisms growing in the form of much branching mycelium, which may break up into segments that function as conidia. Sometimes parasitic, with clubbed ends or radiating threads, conspicuous in lesions in animal body."

In summarizing this case, the following would be the main points of interest: Branching mycelia with sporelike bodies

were found at each and every examination. Active segmentation has been demonstrated. The organisms undoubtedly belong biologically to the class of nocardia. A microscopic section would be very desirable for confirmation of diagnosis.

## REFERENCES.

1. Wright: Jour. of Med. Research, V. XIII, 1905, p. 302.
2. New and Figi: Surg., Gyn. and Obst., V. XXXVII, 1923, 617-625.
3. Bostroem: Beitr. z. path. Anat. u. z. allg. Path., V. IX, 1891, 1-240.
4. Buchanan: General Systematic Bacteriology.

XXVII.

TREATMENT OF PURULENT LEPTOMENINGITIS  
SECONDARY TO OTITIS MEDIA.\*

By J. E. McASKILL, M. D.,

WATERTOWN, N. Y.

A diffuse leptomeningitis is probably the most dread disease that complicates a purulent otitis media, and any form of therapy that offers assistance in the treatment of this disease should be considered carefully.

Circumscribed pachymeningitis has been reported cured many times, but in these cases the infection is well limited by firm adhesions. This condition usually occurs where there is a fistulous perforation of the dura adjacent to an extradural abscess. By removing a large area of the necrotic inner table of bone, after exenterating the mastoid cells, and then splitting the dura to establish good drainage the infection very often clears.

Diffuse leptomeningitis most frequently has resulted through extension of a labyrinthine suppuration by way of the internal meatus. The inflammatory process usually quickly spreads through the subarachnoid space to the base of the brain, upwards over the cerebral hemispheres and down the cord. These cases usually rapidly progress to a fatal termination. When one examines the brain and meninges of the cases that come to necropsy and notes the general accumulation of purulent fibrinous exudate, in some places so dense that the vessels and convolutions are concealed and the ventricles filled with purulent fluid, one realizes that surgical procedure alone could have very little effect on so widespread an infection.

It does seem that if the subarachnoid space could be drained freely and a nontoxic and nonirritating germicidal fluid introduced in the early stages of the disease, something might be done to stem the infection. The results with antimeningo-

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\*Candidate's thesis, American Laryngological, Rhinological and Otological Society.

coccus serum, when injected into the subarachnoid space in patients suffering from epidemic meningitis, have been so satisfactory that this serum is employed almost universally.

Typing the organisms derived from the spinal fluid and injecting type sera homologous to the organism found has not proved very successful, probably because so many types shading into each other are possible.

Disinfecting the subarachnoid space by giving large doses of urotropin has been tried, but no satisfactory results have been obtained.

In 1920, Dr. James B. Ayer of Boston reported the results of his experiments, which showed that there was a balance between the cerebrospinal fluid pressure and the cerebral venous pressure. If a certain amount of cerebrospinal fluid were withdrawn, either by a cisterna magna or lumbar puncture, and then an intravenous injection given of a solution containing selected organisms a meningitis could be produced. These organisms passed from the blood stream to the cerebrospinal fluid, owing to the lowered pressure of the latter. In like manner, if a drug containing a dye were injected intravenously after a considerable amount of cerebrospinal fluid had been withdrawn, it was demonstrated that the dye quickly found its way to the cerebrospinal fluid. It therefore seems possible that injecting intravenously some nontoxic germicidal fluid after withdrawing spinal fluid should be of benefit in the early stages of a purulent meningitis in checking the infection. Injections should also be made directly into the subarachnoid space, preferably by way of the cisterna magna, in order to obtain a higher concentration of the germicidal fluid.

The cisterna magna is really an expanded portion of the subarachnoid space, and communicates freely with the other cisterns and with the narrow channels on the surface of the brain. It is continuous below, through the foramen magnum, with the posterior part of the subarachnoid space of the cord, and in front with the cisterna pontis, which communicates with the anterior space of the cord. In the dorsal and lumbar regions the septa dividing the subarachnoid space are fairly complete. Thus it would seem that the most advantageous point to inject a germicidal fluid into the subarachnoid space would be by way of the cisterna magna. Ayer has pointed out that it has

the strategic position as distributing center of the spinal fluid to both the cerebral and the spinal subarachnoid space. Also, since meningitis is in the beginning a cerebral infection, the nearer the site of the disease the injection is made the greater will be the concentration of the germicidal fluid, which should give a better therapeutic result than fluid injected into the lumbar subdural space.

Dr. Franklin Ebaugh of Denver reported in the A. M. A. (Vol. 85, No. 3, 1925) that in performing approximately 1,550 punctures of the cisterna magna in a total of 190 patients no serious accident occurred.

Numerous antiseptic fluids have been given intravenously and intraspinaly in animal experimentation to discover one that could be given in a germicidal strength without causing severe toxic symptoms. Optochin has been used with some success, but has been found to be effective only against a pneumococcus infection. Fleischmann has reported one cure of a suppurative meningitis by the repeated intravenous injections of trypanflavin after withdrawing cerebrospinal fluid. He noted after the second injection that a lumbar puncture showed the cerebrospinal fluid to be distinctly yellow (Klin. Wchnschr., 1922, 1, 217-220).

Dr. George Draper, New York, found the treatment of cerebrosyphilitics by intravenous and intraspinal therapy very effective in a certain type of case (Amer. J. of Med. Sc., Vol. CLXIX, No. 1, 1925).

Young, Hill and Scott of Baltimore reported in the Archives of Surgery (May, 1925, Vol. 10), their experiences with mercurochrome—220 soluble, in a series of 210 cases, and were convinced that the drug could be introduced into the blood stream or cerebrospinal fluid with safety if carefully given. Some other observers have not been able to corroborate entirely these findings and feel that the drug should be given only in desperate cases. The drug contains mercury, and a few cases of mercurial poisoning have been reported.

I have used mercurochrome quite freely in the ear, nose and throat and in open wounds for the past three years, without noting any symptoms of mercurial poisoning beyond a mild stomatitis. I have injected a 1 per cent solution intravenously and intraspinaly a number of times during the past two

years, and while in a few cases there was a quite severe gastrointestinal reaction and evidence of irritation to the urinary organs, these conditions were usually transient.

During the past year I have had the opportunity of administering this drug in three cases of purulent meningitis, and wish to report one recovery. Fortunately, treatment was commenced in this case during the early stage. It would seem that the most important consideration is the early recognition of the disease and to start the treatment without delay before the condition progresses to a cerebritis, at which stage it is too late for any treatment to be effective.

M. Y., age 11, was admitted to hospital with the following history:

Four weeks ago had severe pain in left ear. Purulent discharge commenced forty-eight hours later. Discharge continued for one week and then ceased gradually. The ear has remained dry and free from pain since. Three days ago the patient commenced to have headache and frequent attacks of vomiting and dizziness. Day of admission headache became severe and vomiting of a violent nature occurred every few minutes. Internal strabismus developed in the left eye. Family physician advised removal to hospital. I examined the child on admission and found the following symptoms: Patient appeared extremely ill. Temperature 105, pulse 130. Was roused from a comatose condition with difficulty and was then very irritable and complained of headache and pain in the back. Pupils were dilated and photophobia was marked. There was paralysis of the left external rectus with the resultant internal strabismus. The neck and extremities were quite rigid and Kernig's sign was positive. Babinski's sign was doubtful. The spine was so extremely tender on percussion that it caused the child to scream out. Examination of the ears showed the right to be normal. The left, which discharged two weeks ago, now appeared almost normal. The perforation had closed, the landmarks were clearly visible and the drum membrane gray in color. There was no sagging of the canal wall. There was, however, total loss of hearing. An X-ray examination of the mastoids showed the cells of both sides to be clear cut. It was thought from the history and findings that the infection had spread by way of the labyrinth. After consultation with

Dr. M. M. Gardner, a lumbar puncture was done and about 20 c. c. of a turbid fluid under considerable pressure was withdrawn slowly. A bedside smear showed a Gram positive organism. A few minutes later, after taking a blood culture, 20 c. c. of mercurochrome—220 soluble, in a 1 per cent solution, made from the scales (the tablets should not be used) and freshly distilled water was injected intravenously. There was a slight gastrointestinal reaction. The vomitus, stools and urine were stained pink. The temperature, which was 105, dropped during the next ten hours to 101, but 24 hours later rose to 106, and the patient's general condition became much worse with deep coma. The rigidity was more marked. There was twitching of the muscles, and an examination of the eye grounds showed both discs to be swollen. The cisterna magna was then punctured, using the technic as outlined by Ayer (*Amer. J. Med. Soc.*, 1919, CLVII, 789), and 30 c. c. of a turbid fluid was withdrawn slowly and 20 c. c. of a 1 per cent solution of mercurochrome at body temperature was injected by the gravity method. The pulse and respirations were watched closely. There was only a slight gastrointestinal reaction. The temperature, which was 106, dropped to 96 in 24 hours. The change in the patient's condition was amazing. Questions were answered intelligently and nourishment taken easily. During the next four days the temperature fluctuated between 99 and 101, and the general condition continued to improve. During this period the subarachnoid space was drained three times and the mercurochrome solution injected. One injection was given in the lumbar region. The patient had a rather prolonged convalescence, due to a cystitis, which developed later and which was thought due to the necessity for frequent catheterizations. The patient was discharged cured and has remained quite well.

It was believed that the injections of mercurochrome brought the patient out of a very desperate condition, one in which her chances of recovery were practically nil.

While recorded experiences are still too few to warrant sweeping deductions, the drug does seem to be a powerful and relatively safe intravenous and intraspinal germicide.

The following fundamental principles should be kept in mind in order to secure the best results:



1. The drug should be injected as early as possible in the course of the disease.

2. It should be injected at frequent intervals to keep the germicide at a high concentration, since the chief action of the drug is to destroy organisms in the subarachnoid space.

3. The treatment should not be discontinued until the cerebrospinal fluid is free from organisms.

4. If the patient's condition continues to grow worse the administration of the drug into the ventricles, through the open frontal or a trephine opening is indicated, for there is everything to gain and nothing to lose.

5. The injection should always be 5 to 10 c. c. less than the amount of fluid withdrawn, remembering that the brain and cord are placed in a closed cavity and one can cause increased pressure easily.

It is hoped that the experiments in progress now in a number of our institutions will offer the opportunity to produce better results in the treatment of this usually fatal disease.

XXVIII.

CASE REPORT OF ACUTE FRONTAL SINUSITIS  
WITH COMPLICATIONS.\*

By D. A. VANDERHOOF, M. D.,

COLORADO SPRINGS.

Personal history: M. B., age 21, was seen June 12, 1925, for severe pain over and around the right frontal. He had just returned from a trip up Pike's Peak, after having completed a motor trip the previous day from Mason, Texas. Morphine, gr.  $\frac{1}{4}$ , with atropine, gr.  $\frac{1}{150}$ , that was given by a physician the previous day, gave only slight relief from pain. A small amount of adrenalin chlorid was used in the nose, hot packs were applied to the frontal area, and continuous inhalations of compound tincture of benzoin and spirits of camphor were used, but without relief. At the time I first saw him, there was considerable edema of the right eyelid, extending up to the margin of the hair and around toward the right temple. This region was all slightly tender. There was a profuse discharge of pus from the right nostril, and with suction a large amount of pus was withdrawn. He was sent to the hospital for further observation and treatment. He entered Bethel Hospital June 13th. At that time he had a slight headache over the right eye and right temporal region, also some occlusion of the nasal passage on the same side, with quite profuse nasal discharge.

Past history as given at that time was unimportant.

Physical examination: White, male, age 21. Well developed and nourished. Lies in bed complaining of severe frontal and right temporal headache.

Head: Scalp is negative, except for tenderness over upper portion of frontal bone and extreme tenderness at the medial portion of the right orbital ridge and also over the right nasal bone. There is moderate tenderness over the right temporal area.

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\*Read before Midwestern Section, American Laryngological, Rhinological and Otological Society, Omaha, Neb., February 11, 1926.

Eyes: There is slight edema of the right eyelid, but the eye can be opened slightly. There is a moderate mild conjunctivitis of the right eye. Cornea and anterior chamber negative. Pupils react to light and accommodation. Ocular pressure is normal. There is no exophthalmos. Vision is normal. Has no scotoma or diplopia.

Ears: Negative.

Nose: There is considerable swelling of the right nostril, with a small amount of pus coming from the region of the right frontal. Left nostril is normal.

Mouth: Lips are normal. Teeth in good condition. Tongue is moderately coated and dry.

Throat: Pharynx, tonsillar crypts and pharyngeal mucous membrane are negative.

Chest: Examination of lungs, heart and blood vessels negative. Back negative.

Abdomen: Rounded. No tenderness, rigidity or spasm.

G. U.: Negative. Extremities negative.

Urinalysis: On entrance was as follows: Specific gravity 1.028, color amber, reaction slightly acid, albumin faint trace, sugar negative, and sediment few pus cells and occasional casts.

June 14, 1925. High mark of temperature was 102 at 11 a. m. Pulse 80. Has only a little pain over frontal, but the right eyelid is very edematous, and there is slight congestion of the ocular conjunctiva. Hot nasal irrigations were used every two hours, also hot packs over right frontal and right side of head. Suction was used both morning and afternoon, and a considerable amount of pus obtained. Has been very bright today and fairly comfortable. Advised intranasal operation, right side, for promoting better drainage. This was refused.

June 15, 1925. Found patient this morning to have considerable swelling of the subcutaneous tissues of the forehead, right eye and cheek. Also slight swelling of the left eyelid. External operation on the right frontal was advised at once. This was refused, but he would now permit an intranasal operation to promote freer drainage. Anterior end of middle turbinate, right side, was removed, but no more done, as bleeding was so profuse and packing for same was contraindicated. Pulse and temperature were about the same as yesterday, but

he was having pain so severe that it was necessary to resort to codein, gr.  $\frac{1}{2}$ , which gave him relief.

June 16, 1925. Swelling about the same today, and there seems to be no edema of the left eyelid. Edema now extends back a couple of inches into the hair. There is some swelling of right side of neck under the jaw. This swelling extends into the anterior angle of right side of neck. Swelling around the right eye is not as red as it was. Right nostril is draining freely, and suction was used twice today with good results. Mind is clear and he says that he feels good, though general condition appears the same. External operation again insisted upon but refused. Some fluctuation can now be elicited on the forehead above the right eye. Pus from nose at this time showed by smear: Gram positive micrococci frequent, while the culture showed a growth of staphylococcus albus. X-ray examination of the sinuses showed marked density of all sinuses on the right side. The left frontal and anterior ethmoids showed less marked involvement.

Dr. Frank Dennis of Colorado Springs was called in consultation and his report was as follows: Large amount of purulent discharge from right nostril, landmarks in nose obscured by swelling. Pus apparently comes from middle meatus. There is extensive edema involving the right eyelid, bridge of nose, and to some extent the left eyelid. This edema extends up over the forehead and over the vertex almost to the midline, also down the side of the right temple and somewhat below the right eye. There was a tender point in the roof of the right orbit.

Consultant's diagnosis: Acute sinusitis of the anterior group on the right side with probable rupture through the anterior wall of the right frontal sinus.

Consultant's recommendation: Exploratory operation and drainage of right frontal sinus.

June 17, 1925. Swelling over the face was a little better this morning. About 8 a. m. he had a very severe pain over the left eye, which lasted about two hours. About 10 a. m. he said that there was suddenly a crackling noise in his head, followed by a free discharge of blood from the left nostril. Pain was relieved at once. He consented to be operated on the follow-

ing morning, as he thought his father would arrive by that time. Blood examination at this time showed 14,400 white cells, 90 per cent polymorphonuclears, 3 per cent small lymphocytes and 7 per cent large lymphocytes.

Dr. E. M. Marbourg, oculist, was called in consultation, and his report is as follows: Marked congestion of retinal veins, more so in right than left eye. Some proptosis of the right eye. Lids and conjunctiva of the right eye edematous. Lids of left eye somewhat less edematous than right. Right pupil reacts sluggishly, with inclination to be contracted. Ptosis marked in right lid, left lid to less extent. Anesthesia marked right cornea. Left cornea normal. Pain on pressure over right mastoid.

Probable diagnosis: Cavernous sinus thrombosis, involving right side more than left. Slight involvement of left side.

June 18, 1925. General condition about the same. There is now a little edema back of the right ear and considerable pain in temple. Can open eyes a little better, but the conjunctiva of the right eye is a little more edematous. Swelling on forehead seems a little less. Nasal discharge about the same. Highest temperature today 104, pulse 85 and respirations 22.

Dr. Frank Dennis saw the patient again today and made the following report: When seen today the edema of the right eyelid was slightly less, although the swelling of the conjunctiva in the right eye was greater. Edema of the left eye was slightly less. The tenderness had extended up the forehead to well beyond the hair line, and the edema on the right side of the head also extended downward behind the mastoid region. Washings from the right antrum revealed considerable pus. Patient was brought to the operating room at 4:30 p. m. and anesthetic of ether was given. Incision was made from a little to the right of center of forehead, beginning at the hair line and extending to about the center of the nose. Tissues very edematous. There was a free flow of pus in the incision, which was made down to the bone of the forehead. Pus had burrowed on the right side to the zygomatic process and half way over to the zygomatic process on the left side. This was all elevated by curved forceps and the region thoroughly cleaned. No roughened bone was found, nor could any place

be found where the pus had ruptured through from the frontal sinus. Opening was quickly made into the frontal sinus, and a considerable amount of thick yellow pus was found. Four drainage tubes were inserted under the periosteum of the forehead, and one tube inserted into the frontal sinus. No sutures were used and skin around the wound was thoroughly covered with vaselin gauze. An irrigation of Dakin's solution was ordered every two hours.

June 19, 1925. Condition was good this morning. Dressings were changed. There was less swelling of the right eye, head less tender, and patient seemed much brighter. Temperature a little lower. High point, 102 8/10; low point, 100 6/10. Pulse was 100 at 8 p. m. Blood culture showed a large growth of staphylococcus albus. Smear from right frontal showed large growth of staphylococcus albus.

June 20, 1926. Says that he feels very good this morning. Can open both eyes better. Almost no discharge from nose. There is some fluctuation this morning, a little to the right of the top of the head, and also a suspicious area to the right of the occiput, with more edema over the right mastoid region. Pulse running about 100, with highest point in temperature 103 6/10 and low 99. Has had no chills and is not feeling bad. Urinalysis as follows: Specific gravity 1020, color amber, reaction slightly acid, albumin faint trace, sugar negative, and sediment epithelia few, pus few, and occasional casts not seen. At 8 p. m., Dr. M. O. Shivers, general surgeon, made a number of scalp incisions down to the bone, and pus was found everywhere except in the incision over the mastoid. Periosteum elevated and parts thoroughly cleaned and packed.

June 21 and 22, 1925. Condition about the same these two days. Dressings changed every day and kept moist with 50 per cent alcohol. Highest temperature during these two days was on the 21st, which was 103 4/10; lowest on the 22d, which was 101. Urinalysis was as follows: Specific gravity 1,026, color amber, reaction acid, albumin faint trace, sugar negative, and sediment epithelia few, pus few, occasional blood cells and few hyalin casts. Blood examination was as follows: Hemoglobin 75 per cent, red blood cells per c. c., 3,400,000, color index 1.1 per cent, white cells 14,200, polymorphonuclears 93

per cent, small lymphocytes 3 per cent, large lymphocytes 1 per cent and transitionals 3 per cent. During the last two days he has been very restless and nervous. Murphy drip has been given for the last three days, but today was discontinued, as he was unable to retain it. Regular treatment of eyes, dressings, etc., were continued as previously done. Passing normal amount of urine. Given soft diet. Water being pushed.

June 23, 1925. Somewhat irrational at times. Temperature 104 8/10 axillary at 7 p. m. No chills and says that he does not feel bad. Ate fairly well. Dressings changed and found wounds very dry. Pulse 110 to 120. Respirations 20 to 35. Strychnin, gr. 1/30, every six hours. Murphy drip of glucose and soda, continuously, and hot compresses to right eye for ten minutes out of every hour. Some swelling of left wrist and swelling over the knuckle of the right index finger. Feels as if there was some fluctuation at this point. Hot compresses applied. Very restless and hard to restrain. Takes plenty of water and some food by mouth.

June 24, 1925. Temperature remains high today. The highest temperature was 105 axillary, pulse 120 and respirations 44. At 9:40 a. m., 10 c. c. of mercurochrome was given intravenously by Dr. Gydeson. This was repeated at 5:10 p. m. Normal salt solution, O 2, by hypodermoclysis, was given twice during the day. Strychnin and other treatments were continued. Spirits frumenti, drams 2, in eggnog, has been given t. i. d. during the last three days. Blood examination was as follows: Hemoglobin 70 per cent, red cells per c. c. 3,900,000, color index .9 per cent, white cells per c. c. 19,200, polymorphonuclears 88 per cent, small lymphocytes 7 per cent, large lymphocytes 3 per cent, and large mononuclears 2 per cent. Scalp wounds were dry. No pus in frontal sinus. No pus in nose. Extreme ptosis of the right eye. Chest report negative. Abdomen rather tense. Incontinence of feces and urine. Definite abscess on the right middle carpal joint.

June 25, 1925. Failing rapidly today. Temperature 106 6/10 axillary, pulse 150 plus, and respirations between 40 and 50. Mercurochrome, 15 c. c., given at 9:30 a. m., and 25 c. c. given at 5:30 p. m. Normal salt solution was given as the day previous. Camphor in oil, 1 c. c., and digitalin, 1/100 gr., were

alternated every two hours. No results from mercurochrome. Blood examination was as follows: Hemoglobin 68 per cent, red cells per c. c. 3,400,000, color index 1 per cent, white cells per c. c. 19,000, polymorphonuclears 89 per cent, small lymphocytes 4 per cent, large lymphocytes 6 per cent and transitionals 1 per cent. Urinalysis was as follows: Specific gravity, 1.016, color amber, reaction slightly acid, albumin faint trace, sugar negative, epithelia few, leucocytes few, red blood cells occasional, and no casts found.

June 26, 1925. Expired at 11:10 a. m.

Postmortem is as follows: The rigor mortis is extreme. No postmortem lividity. The skin has a peculiar scarlatinal rash. There is a vertical incision three inches long over his forehead and other incisions on the right side of the cranium. There is a fluctuation abscess over the third metacarpal phalangeal joint right. There is a small area of fluctuation over the left carpal area.

The thorax was opened by regular incision. Breast plate removed. The left lung was covered by a thick yellow and hemorrhagic exudate. By this exudate the lung was adherent to the chest wall and pericardium. About 300 c. c. of fluid was found behind this lung. The right pleural cavity had a similar yellowish and hemorrhagic plastic adhesion. The left lung was removed. The bronchials excreted considerable frothy material. On section about two ounces of fluid was found to be incapsulated between the superior and inferior lobes. On section the lung was gray, the vessels standing out as dark red areas. It was crepitant but had a marginal hepaticization. The right lung same.

Pericardium: There was a thick plastic exudate on the pericardium. The parietal surface of the pericardium was smooth, but had many small petechial hemorrhages on the surface. There was a normal amount of straw colored fluid in the sac. The serous pericardium was negative.

No measurements were taken of the heart, but it seemed to be slightly smaller than normal in size. Heart muscles were normal color and of good structure. The endothelium and valves were negative. The ascending aorta was negative.



Abdomen was opened. No excess of fluid. Liver was slightly larger than normal size. Gall bladder negative and emptied easily. On section liver showed chronic passive congestion. No abscesses were found. No subphrenic abscess was found.

The spleen was enlarged times three. A gray fibrous area was found on its lateral surface. On section it bled freely and was very dark in color.

The right kidney was enlarged, bluish black in color, with small hemorrhagic areas on its surface. On cut section it bled freely, dark venous blood. The vessels were filled with dark blood, but no thrombi could be found in the portal vein.

The left kidney was enlarged and capsule stripped easily. It was a peculiar red color, probably due to mercurochrome.

The pelvis and calyces were negative. No examination of the stomach, duodenum, small or large intestines, ureters or bladder was made. The abscess on the hand was not opened.

Head: Scalp and aponeurosis removed in the usual manner. Pus was found beneath the incised area on the right side of the head. The cranium was removed in the usual manner. A thin layer of a tenacious creamy pus was found over the right frontal area and another collection over both parietal areas. A definite infiltration was found along the middle branch of the left meningeal artery. The dura mater was removed. Beneath the dura mater, over the extreme point of the right frontal lobe, a small abscess was found to have compressed that portion of the right frontal lobe. The cerebrum was removed intact and sectioned and no pus found. The tentorium was incised and cerebellum removed. The cerebellum was sectioned and many small thrombotic areas were found in the right side. The rest of the dura mater was stripped from the base of the skull and along the basilar portion of the right meningeal artery. A small collection of pus was found at the base of the skull. No internal rupture of the frontal sinus was observed.

The roof of the orbit over the right cavernous sinus was removed and a small amount of pus was obtained. On opening the roof of the right orbital cavity about 10 c. c. of sanguinous fluid escaped. The rest of the roof of the orbital cavity was

removed, and by probing about in the postorbital fat no pus was obtained. The left orbital cavity and cavernous sinus were explored but nothing was found.

Anatomic diagnosis: 1. Frontal sinusitis with external perforation. 2. Subaponeurotic abscess. 3. Extradural abscess. 4. Subdural abscess over right frontal lobe. 5. Abscess of right cavernous sinus. 6. Multiple thrombi in right half of cerebellum. 7. Acute fibrinous pleurisy. 8. Right kidney, acute passive congestion with thrombi formation. 9. Liver, acute passive congestion. Postmortem by Dr. C. S. Gydesen.

600 EXCHANGE NATIONAL BANK BLDG.

XXIX.

SIX CASES OF DEFINITE MASTOIDITIS IN WHICH  
THE MIDDLE EAR WAS DEFINITELY NOT  
AFFECTED.

BY B. E. HEMPSTEAD, M. D.,

MAYO CLINIC,

ROCHESTER, MINN.

Six cases of definite mastoiditis, in which the middle ear apparently was not affected, have been observed in the Mayo Clinic. The incidence of this condition is low as compared with more than 500 cases in which the middle ear was obviously affected during the same period. The term primary mastoiditis, as it has been designated in the literature, is misleading in such cases since the infection must be primary in the mastoid cells, and not secondary to inflammation of the middle ear that has cleared up. It is possible to conceive of a blood borne infection, after a severe local injury, as in osteomyelitis of other bones, although there is no more reason to expect such an infection in the mastoid than in other cavities, for example, the accessory sinuses.

REVIEW OF THE LITERATURE.

In 1915, Dabney reviewed the literature on this subject. He classified the condition as idiopathic mastoid abscess. He says, "I wish the title to be understood as meaning an abscess in the mastoid process of the temporal bone without any immediately preceding or accompanying inflammatory involvement of the tympanum. It will be observed that the definition has been made particularly restrictive, in that it specifies merely inflammatory involvement, not abscess of the middle ear, as there are many cases in which it would be difficult to demonstrate an actual abscess and yet the mastoid disease would be plainly an extension of the tympanic inflammation."

Dabney reported twenty-four cases, to which he added two of his own. He argues that infection may pass directly through

the middle ear without setting up inflammation, and cites as an example of this process tuberculous lymph nodes in the neck, which had been infected from the tonsils, although no evidence of infection remained in them.

It is generally agreed that the infection in cases of mastoiditis comes from the nasopharynx by way of the eustachian tube. The middle ear may be only slightly involved. Access to the mastoid cells is gained through the aditus ad antrum and, if this passage is small, it will soon be sealed off, leaving no means of drainage for the infected cells, whereas, if the infection is in the middle ear, there may be sufficient drainage through the eustachian tube. It is worthy of note that infection may exist in the middle ear without causing pain, discomfort, fullness, tinnitus, or impaired hearing.

Mastoiditis without apparent involvement of the middle ear should not be confused with latent suppurative otitis media. In the latter type of case there is deafness and sometimes pain, but no spontaneous discharge of pus. There may or may not be mastoid involvement, and other complications; the tympanic membrane is without luster, full, and sometimes markedly bulging, and myringotomy is always followed by the discharge of pus, whereas in cases of mastoiditis without apparent involvement of the middle ear, myringotomy is never followed by the discharge of pus.

A careful review of the literature has revealed the reports of thirty-two cases in English and American literature since Dabney's report in 1915. The description of some of the cases is exceedingly meager. In many instances no mention is made of myringotomy, and no definite operative findings are given. It is quite probable that some of these are cases of latent suppurative otitis media instead of mastoiditis without apparent involvement of the middle ear.

Welton, in 1915, reported a case in which there was considerable pain in the middle ear, but all landmarks were present. Broder, in 1915, reported a case of an infant, aged three months, with a subperiosteal abscess. Myringotomy had been performed without obtaining pus. The child had sustained an injury to the mastoid bone one week previously, and it was believed that osteomyelitis had followed. Broder reported a case of a boy, aged seven years, with a subperiosteal abscess.

The appearance of the drum was normal, but there was slight deafness, and operation revealed extensive necrosis. Bonner and Dutrow, in 1915, reported one case in which there was definite involvement of the drum, which, I believe, excludes the possibility that the case was mastoiditis without apparent involvement of the middle ear.

Tomlin, in 1916, reported a case in which the patient had been an invalid for ten years. Because of slight pain back of the ear, the mastoid was opened, and chronic suppurative granulomatous infiltration and inflammation, with thin, dark, straw colored fluid were found. The patient recovered and in a short time gained 20 pounds (9 kg.).

Sonnenschein, in 1917, reported a case of a man, aged thirty-four, who had complained of a cold in the head with pain around and back of the ear. There was no deafness and no change of landmarks. Myringotomy was negative twice. The patient had had a rise in temperature and leucocytes. One month after the first examination, swelling appeared over the mastoid. Operation revealed extensive destruction of cells. Long, in 1917, reported a case of a girl, aged twelve, suffering from severe chills, fever, prostration, and pain in the left ear. Two weeks previously she had had a severe cold in the head; the membrane was red and injected; incision did not disclose pus. At operation, a badly necrosed mastoid with sinus phlebitis was found. Dulaney, in 1918, reported three cases, only one of which could be classified as mastoiditis without apparent involvement of the middle ear.

Knight, in 1919, reported a case of a young girl who had been sick in bed for two weeks with influenza. Three months later, swelling appeared over the mastoid. Operation revealed marked destruction of the cells. Miller, in 1919, reported two cases: one following an attack of measles, during which the child complained of a mild earache, and one following rhinitis. Probably neither of these cases is idiopathic. West, in 1919, reported a case of subperiosteal abscess without involvement of the middle ear. He did not give the findings relative to the middle ear. There had been a daily chill and rise in temperature. The sinus and the dura had been exposed during the operation. The case, however, proved to be one of malaria and not sinus thrombosis. Kerrison, in 1919, reported a case

of a boy, aged four months, without involvement of the middle ear. The child had been fretful previous to the appearance of the subperiosteal abscess. Friesner, in 1919, reported seven cases of mastoiditis with perisinus abscess in which the middle ear had not discharged. These cases were all observed during January and February. The appearance of the membrane and a myringotomy were not recorded.

Glogau, in 1921, reported a case with perisinus and extradural abscess, with operation and recovery. He believes that primary mastoiditis is a distinct clinical entity, and that the infectious material passes through the middle ear without finding favorable conditions for growth. The aditus, being poorly supplied with blood vessels, allows the infection to reach the antrum, and swelling closes off the opening from the middle ear. It is hardly possible, however, that the infection would cause so much reaction in the aditus ad antrum and none in the middle ear. Zamora, in 1921, reported three cases, in one of which there had been severe earache six months previously, and catheterization gave the impression of fluid in the middle ear. The condition of the drum and myringotomy were not mentioned. Mollison, in 1921, reported two cases: one followed an attack of influenza. No information was given with regard to the appearance of the drum, or of a myringotomy. This case may have been latent otitis media. In the second case the patient had had earache ten days previously. Likewise, nothing was said of the appearance of the drum. Both of these patients later had sinus thrombosis, and both recovered. Dawson, in discussing this paper, said that he had had cases in which the middle ear cleared up absolutely, probably as a result of the drainage through the eustachian tube; the mastoid disease, however, went on to abscess. Scott said that he had reported two cases of extradural abscess eleven years previously, in which the hearing was perfect and the membrana tympani normal. Taylor, in 1921, reviewed a case of atypical mastoiditis following staphylococcemia. This was clearly a mastoiditis of blood borne infection, which, it must be admitted, is a rare condition. Ballance, in 1921, reported a case of acute osteomyelitis of the mastoid which had nothing to do with the primary disease. He could see no reason why osteomyelitis should not take place in the temporal bone as

well as in the other bones of the body. He reported a very interesting case. A boy had died from septicemia three weeks after an operation for harelip. The wound, which had healed perfectly, began to break down. There was no rise in temperature, and there were many petechial spots over the body. Necropsy revealed extensive destruction of the petrous bone and mastoid cells, with suppuration in the sinuses. The ears had been examined repeatedly. Hughes, in 1923, reported the case of a boy, aged thirteen, with pain in the right ear and definite swelling over the mastoid. There was a slight discharge which lasted several weeks. Roentgenograms were positive. There was a gradual recovery without operation. Eighteen months later the patient had pain and swelling behind the same ear; there was a slight infection of Shrapnell's membrane and a slight deafness. Operation showed a definite mastoiditis. The author hints that this might have been a lighting up of the old process. Heggie and Knaney, in 1924, reported two cases: one in a little girl, aged twenty months, and the other in a girl, aged three years. There were no signs of trouble in the middle ear. Both had subperiosteal abscesses. In one the invading organism was a staphylococcus and in the other a streptococcus.

Wagers, in 1924, reported a case of an infant, aged seven months, with definite subperiosteal swelling and normal appearing tympanic membrane. Incision of the membrane did not reveal pus, but operation showed definite mastoiditis. Diehl, in 1924, reported a case of a man, aged forty-one, who had complained of a cold in the head. There was no aural pain or discharge, no changes in the drum membrane, but there was a sense of fullness in the right ear. In July, severe pain back of the right ear and slight swelling appeared. Roentgenograms showed destruction of cells. Operation showed a jelly-like substance in the mastoid, together with an epidural abscess. Bacteriologic examination disclosed a pneumococcus. Brown, in 1924, reported the case of a young woman who in July, 1923, went swimming in a plunge. She developed a cold in the head, intense pain in the right ear, and a temperature of  $101^{\circ}$ . Pain continued for one week with no evidence of tympanic trouble in the drum. It lasted until August 6. She began to have headaches September 15. October 6 she went

to bed with a chill and temperature of 103°. Blood was discharged through the external canal wall. The drum membrane still showed no evidence of pathologic changes. At operation the sinus was found exposed and the middle ear sealed off.

#### REPORT OF CASES.

Case 1.—A man, aged twenty, came to the clinic September 20, 1922, because of painful swelling of the right mastoid and upper cervical region. For seven years he had had recurring attacks of furunculosis in both external canals, worse in the right. The furuncles ruptured spontaneously, the discharge lasting from two to seven days. The last attack had occurred five weeks before, with spontaneous rupture in eight days. Swelling over the mastoid had been present for six days. The patient had had night sweats for five weeks.

The hearing was normal. Findings in the nose, nasopharynx, throat, larynx and both tympanic membranes were negative. There was a slight swelling and tenderness in the region of the isthmus and right external canal, and a large tender swelling below the tip of the right mastoid and extending down the sternocleidomastoid muscle. Roentgenograms revealed destruction of cells. The leucocytes numbered 14,600. A tentative diagnosis was made of mastoiditis with Bezold abscess.

The usual mastoid incision was made. Bleeding of the cortex and perforation in the posterior tip were found. All cells were removed. The aditus ad antrum was very small. The abscess cavity was drained. Bacteriologic examination revealed staphylococcus. The tympanic membrane was incised, but pus was not obtained. The patient was dismissed two weeks after the operation, with the wound healed.

Comment.—It is likely that the last attack was otitis media and not furunculosis, and it is barely possible that the mastoiditis was primary, the furunculosis having been the source of infection.

Case 2.—A boy, aged fourteen, was brought to the clinic January 31, 1919, because of swelling behind the right ear of three days' duration. He had had chorea until the age of twelve, and had never been well. He had had influenza two months before, was in bed two weeks, and was confined to



the house four weeks. Soon after the swelling began there was slight pain in the right ear for one hour.

The hearing was normal. There was slight redness and swelling of the external canal and the tip of the mastoid, with tenderness on pressure. The tympanic membrane was not seen. By catheterization of the eustachian tube, the middle ear was shown to be clear. The roentgenogram revealed extensive destruction of the mastoid cells.

At operation marked edema of the subcutaneous tissue and perforation of the cortex of the mastoid over the antrum were found. There was marked destruction of cells, and a parasinus abscess. The antrum was small and located with difficulty. The tympanic membrane was incised, but no pus was obtained.

Comment.—This patient, no doubt, had had otitis media during the attack of influenza; the infection spread to the mastoid, and the inflammation, coincident with the infection, sealed off the aditus ad antrum. Streptococcus was revealed on culture. The mastoid cavity was dry, and the wound healed in ten days. At no time was there discharge from the middle ear.

Case 3.—A man, aged forty-one, came to the clinic August 5, 1922, with pain, redness, and swelling over the left mastoid. About four months before, he had had influenza, and was in bed for three weeks. He had had severe pain in the left ear, followed by a bloody discharge mixed with cerumen. (From the patient's description, the disease may have been myringitis bullosa.) At the end of two weeks there was slight tenderness over the mastoid, but this disappeared in a few days. The patient had remained well until two weeks before, when he developed an acute cold. One week later the tissue over the left mastoid became painful and swollen.

The hearing was normal. Aside from septic tonsils, no abnormality was noted in the nose, throat, nasopharynx, larynx, or tympanic membranes. The posterior superior wall of the canal drooped slightly. There was a large fluctuating mass over the left mastoid region. Roentgenograms revealed destruction of the mastoid cells.

A large subperiosteal abscess and perforation of the cortex on the posterior aspect of the tip were found at operation.

All the cells were broken down and filled with pus and granulation tissue. An effort was made to clear these out. Myringotomy did not reveal pus. The wound healed and the patient was dismissed on the fifteenth day.

Comment.—From the operative findings it would appear that the mastoid infection, which was of low virulence, and aggravated by the severe cold, was simultaneous with the pain in the middle ear.

Case 4.—A woman, aged seventy-five, came to the clinic April 16, 1925, with pain and redness over the right mastoid region. There was no history of ear trouble until after an attack of influenza the previous winter. Since then there had been a sensation of fullness in the ear. Two weeks previous to her admission to the hospital she had had some pain in the ear.

The hearing was normal. Both drum membranes appeared normal. No perforation of the right ear drum and no evidence of any trouble in the middle ear were found. Roentgenograms showed complete destruction of the mastoid cells on the right side.

A subperiosteal abscess presenting at about the middle of the posterior wall of the canal was found at operation; the aditus ad antrum had been completely walled off. The partitions of the mastoid cells were destroyed. The sinus wall was found exposed. The mastoid was completely cleaned out. In ten days the wound was entirely healed. Bacteriologic examination revealed streptococcus mucosus, or Type 3 pneumococcus. The patient is living and in excellent health.

Comment.—As is well known, streptococcus mucosus is very insidious in its attack on the mastoid cells. It is not likely that this infection was introduced by the previous influenza, but it is likely that the infection had been present for some time. This organism would be expected to give just this sort of picture. It does not produce a great amount of pus but it is very destructive in its action.

The following histories of cases of mastoiditis secondary to a very definite previous middle ear infection might have been confused with primary mastoiditis.

Case 5.—A man, aged sixty-two, came to the clinic June 15, 1923, because of pain over the left mastoid region. About

three months previously, he had had influenza followed by bilateral otalgia. Two days after the onset, the right drum ruptured spontaneously; about one week later, the left ear drum also ruptured spontaneously. The right ear had discharged for six weeks, and the left ear had discharged until ten weeks before; it was then dry for seven weeks and discharged for three weeks, before the patient came to the clinic.

The patient was not deaf. There was no perforation to be seen in either drum. A mastoidectomy on the right side had been performed previously elsewhere. No pathologic changes were seen in the left tympanic membrane. The patient had some pain and tenderness over the mastoid process on the left side and the roentgenograms showed considerable destruction of the cells. After two days' observation and treatment the swelling increased over the mastoid area, and a preoperative diagnosis was made of Bezold abscess.

At operation a perforation of the posterior aspect of the tip was revealed. The sinus was found exposed. Most of the pathologic changes were in the tip cells. Bacteriologic examination showed Type 3 pneumococcus. The patient recovered uneventfully and was dismissed in three weeks.

Comment.—In this case the destruction and the insidious work of this organism are seen again. The middle ear had entirely cleared up but extensive destruction had taken place after the closing off of the aditus ad antrum.

Case 6.—A man, aged twenty-one, came to the clinic July 3, 1923, because of pain and swelling in the left postauricular region. About four months previously he had had a severe cold in the head with bilateral otalgia. Both drumheads had been incised, and there was a discharge of pus for four or five days with complete relief from pain. Three months later the patient had a slight otalgia on the left side. For four or five days he had noticed a swelling over the left mastoid region which has become increasingly painful and larger.

The hearing was normal. The tympanic membranes were normal. A large fluctuating mass, 2 cm. in diameter, was found behind the left ear. Roentgenograms showed destruction of the cells.

A large subperiosteal abscess, with rupture of the cortex behind and above the mastoid antrum, was found at operation.

Cell partitions had broken down. Pachymeningitis in the region of Trautman's triangle and also a perisinus abscess in the region of the knee were found. The patient recovered uneventfully and was dismissed three weeks after operation. Bacteriologic examination showed a small gram negative bacillus.

Comment.—This case is very definitely mastoiditis secondary to infection of the middle ear in which the infection had entirely cleared up before the mastoid symptoms became evident.

#### SUMMARY.

It will be recalled that in Cases 2 and 3 there was a history of an antecedent pain in the ear which disappeared without treatment in a very short time (less than an hour in Case 2). The symptom directing attention to the mastoid was the swelling over the mastoid process. The indolent type of infection, followed by the formation of the subperiosteal abscess, could explain the absence of pain as a prominent symptom. The anatomic conformation of the mastoid process may have been a factor, because the aditus ad antrum was very small. It seems evident that these cases could not be grouped in those of blood borne infection. In all cases the structures of the ear and the tympanic membranes were negative on physical examination. In Case 2 a marked fullness of the superior wall of the canal externally to the isthmus was noted. One usually expects to encounter drooping beyond the isthmus.

While the study of these four cases of mastoiditis without apparent involvement of the middle ear does not permit definite conclusions, it indicates the existence of an antecedent otitis media without symptoms. The roentgen ray examination is important. The predominance of the streptococcus mucosus in these cases warns us of the danger in this type of infection and puts us on our guard when the organism is found in the acutely discharging ear.

#### BIBLIOGRAPHY.

1. Ballance: Quoted by Mollison.
2. Bonner, H., and Dutrow, H. V.: Report of a Case of Primary Mastoiditis. *Laryngoscope*, 1915, xxv, 244-245.
3. Broder, C. B.: Primary Mastoiditis: Report of Two Cases. *Med. Rec.*, 1915, lxxxviii, 482-483.

4. Brown, J. M.: Atypical Mastoiditis. Case Report. *Laryngoscope*, 1924, xxxiv, 644-646.
5. Dabney, V.: Idiopathic Mastoid Abscess. *Jour. Am. Med. Assn.*, 1915, lxxv, 501-504.
6. Diehl, J. E.: Report of a Case of Mastoiditis, Epidural Abscess With Apparently No Middle Ear Involvement. *Virginia Med. Month.*, 1924, li, 223-225.
7. Dulaney, O.: Some Clinical Observations on Primary Infection of the Mastoid, With Report of Cases. *Jour. Tennessee State Med. Assn.*, 1918-1919, xi, 264-269.
8. Friesner, I.: Quoted by Knight.
9. Glogau, O.: Primary Mastoiditis With Perisinus and Extradural Abscess; Operation; Recovery. *Laryngoscope*, 1921, xxxi, 318-320.
10. Heggie, N. M., and Knauer, W. J.: Mastoiditis Without Apparent Involvement of the Middle Ear. *Jour. Am. Med. Assn.*, 1924, lxxxii, 1044-1045.
11. Hughes, T. E.: An Unusual Case of Mastoiditis; Operation and Recovery. *Virginia Med. Month.*, 1923, xlix, 726-727.
12. Kerrison, P. D.: Mastoiditis Abscess Without Aural Discharge. *Ann. Otol. Rhinol. and Laryngol.*, 1919, xxviii, 949-951.
13. Knight, F. H.: Case of Perisinus Abscess Without Involvement of the Middle Ear. *Laryngoscope*, 1919, xxix, 433-438.
14. Long, C. H.: Acute Suppurating Mastoiditis Without Tympanitis; Perisinus Abscess, Phlebitis, Streptococcemia, Operation, Recovery. *Ann. Otol. Rhinol. and Laryngol.*, 1917, xxvi, 432-435.
15. Miller, C. M.: Mastoid Abscess Without Suppuration in Tympanic Cavity: Report of Two Cases. *South. Med. Jour.*, 1919, xii, 105-106.
16. Mollison, W. M.: Two Cases of Lateral Sinus Thrombosis Without Otorrhea. *Proc. Roy. Soc. Med., Sect. Otol.*, 1921, xiv, 42-44.
17. Sonnenschein, R.: Primary Mastoiditis, With Report of a Case. *Illinois Med. Jour.*, 1917, xxxii, 167-169.
18. Taylor, H. M.: An Atypical Mastoiditis Following a Staphylococcemia. *South. Med. Jour.*, 1921, xiv, 167-170.
19. Tomlin, W. S.: Report of a Case of Suppurative Mastoiditis Without Tympanitis. *Indianapolis Med. Jour.*, 1916, xxx, 95-97.
20. Wagers, A. J.: Acute Mastoiditis, Apparently Primary, In An Infant Seven Months of Age. *Laryngoscope*, 1924, xxxiv, 453-456.
21. Welton, C. B.: Mastoiditis and Mastoid Abscess Without Suppuration From the Middle Ear and Without Any Apparent Middle Ear Inflammation. *Jour. Ophth. and Otolaryngol.*, 1915, ix, 86-91.
22. West, L. N.: Mastoiditis Without Involvement of the Middle Ear, Complicated by Malaria. *South. Med. Jour.*, 1919, xii, 339.
23. Zamora, A. M.: Mastoiditis and Complications Without Otorrhea, and Some Observations Upon the Resolution of Mastoid Infections. *Guy's Hosp. Rep.*, 1921, lxxi, 216-221.

XXX.

VINCENT'S ANGINA.\*

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History.—In the year 1898, H. Vincent, an army surgeon serving with the French troops in Africa, presented to the Société médicale des hôpitaux de Paris, an account of "an especial form of diphtheritic angina due to a fusiform bacillus," stating that he had observed fourteen cases of this affection during the space of a few years. He felt certain that the disease was much more common than his investigations indicated, and as the causal agent closely resembled that of diphtheria, he believed that the condition was often confounded with diphtheria, and for that reason felt that a complete clinical description, and a report of his observations upon the fusiform bacillus to which it was due, would be of practical value to the medical profession at large.

Two years before Vincent had published the results of his studies as to the etiology of "hospital gangrene," which, thanks to the discoveries of Pasteur and Lister, had long since become a medical curiosity, and was only observed under exceptional conditions of filth and surgical carelessness, such as he had encountered in his campaigns in Africa. He now sought to establish that the causal agent of the disease he had observed, and that of the putrefaction of wounds, once the greatest horror of the army surgeon, were closely related, if not, indeed, identical, and to this end he devoted years of painstaking labor and minute investigation, which, after the lapse of a great deal of time, was acknowledged by the attachment of his name to the malady he was the first to describe clearly.

Vincent did not claim that the condition he was describing, or the bacillus to which he correctly attributed its occurrence, was hitherto entirely unknown to the medical world. As far back as 1883, a fusiform bacillus and spirochete had

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\*Thesis accepted by the American Laryngological, Rhinological and Otological Society.

been detected by an earlier student of bacteriology, Willoughby Miller, an American dentist, working in Germany, in the course of studies made on the bacteria of the oral cavity, of which he gave so complete a description that no doubt can be entertained as to its identity with that now designated by Vincent's name. Miller also noted that the organisms would not grow upon any of the culture media which he employed. In 1894, Plaut, another German investigator, reported two cases of an ulceromembranous angina, and described fusiform bacilli and spirochetes recovered from the ulcerated surfaces, which he designated as Miller's organisms. Plaut found these organisms also in normal mouths, and was able to demonstrate their presence in large numbers where the teeth and gums were in a neglected and diseased condition, though the ulceromembranous condition was entirely absent. In his notes before the Paris Société des Hôpitaux, Vincent expressly mentions that it was the publication about six weeks before of a clinical description of the disease by Bernheim, another German, which moved him to make public the results of the work he had been carrying on for several years.

There is, then, some ground for the German contention that Vincent was not the discoverer of the organism which now bears his name, and for designating it—as is actually often done—as the "Plaut-Vincent bacillus," but even if strict priority should be accorded to Plaut—which is very doubtful at best—certainly the years of patient labor which Vincent spent in making a knowledge of this disease producer available to the medical profession are deserving of the tardy recognition which has been accorded them.

After 1898 the literature on Vincent's angina became very extensive. The Surgeon General's Index, published in 1913, contains no less than 266 entries under the head of "Ulceromembranous Inflammation of the Throat" (Vincent's angina), eighteen of which are under Vincent's own name. For the first few years of the present century the interest in the subject seems to have been quite extended, but later on this interest declined, only to undergo a marked revival when, in 1911, the possibility of successfully combating the disease by the same measures which had proved so efficacious in the treatment of the spirochete of syphilis was first suggested by

Achard. As Sebenq has put it: "The story of Vincent's angina regained actuality in the same moment when we learned of arsenobenzol and the possibility of its employment in spirillosis or spirochetosis. This disease, which in 1904 appeared to be permanently classified with a standardized treatment, suddenly saw its whole history reviewed and rewritten, not alone in respect to an additional chapter on treatment, but even being equipped with an entirely new pathology. Indeed, it has been demonstrated beyond a doubt that it is the spirilla in the fusospirillary symbiosis which is the causal agent in Vincent's angina, and not—as Vincent himself supposed—the fusiform bacillus, and this has been demonstrated by the extraordinary efficacy which arsenobenzol therapy has had upon the disease. It was Achard who, in 1911, was the first to apply this treatment, with a success which has since been duplicated on all sides, so that it is now the therapy which is universally employed."

While Sebenq's last statement may be true for France, I feel that for the United States at least it must be somewhat qualified, and my chief purpose in selecting this subject for presentation to this society is to emphasize the prevalence of this malady, and to direct the attention of physicians to the practically specific treatment which, though easily available to all, is by no means so "universally employed" as the French enthusiast would have us believe.

In this country at least, there seems to have been little general interest in Vincent's angina until the United States entered the European war. English and French medical officers had already observed a very high incidence of an ulcerative stomatitis among the troops under their care. Barker and Miller state that the disease broke out sporadically, was usually preceded by a sore throat, and became so common that it constituted, according to Bouty, 23 per cent of all throat infections. It was soon found that the lesions were invariably associated with a mixed invasion of fusiform bacilli and spirochetes, and the condition was familiarly referred to as "trench mouth," "trench throat" or "trench gums." Probably the condition has no association with trench life as such, but its dissemination is favored by the collection in camps of large numbers of young men. That it may be very infectious in



type was shown by the occurrence of more than 200 cases within two days among 800 prisoners in one of the German camps.

As Cocks has remarked, the number of cases of Vincent's angina encountered in the course of a year depends to a great extent on whether the physician is on the lookout for this particular disease. In his opinion, the fact that the annual report of "one of the large institutions of New York City" listed over 7,000 cases of diseases of the pharynx and nasopharynx as having been treated in 1912, of which but three were recognized as Vincent's angina, "looks like carelessness on the part of the examining physicians."

It would seem, despite all that has been said and written regarding this malady, that, in general, the average practitioner does not keep it in mind, and that its clinical picture so resembles that of diphtheria or the secondary lesions of syphilis that it more often than not is allowed to pass without recognition. Yet the differentiation should not be difficult.

#### BACTERIOLOGY.

According to Barker and Miller, the examination of smears made directly from an ulcer always shows a great variety of organisms, but two in particular tend to predominate. These are:

1. The bacillus fusiformis, first noted by Vincent in cases of hospital gangrene. The fusiform bacillus is from four to fourteen micra long and about .5 micron thick at the center, from which it tapers to the ends. It stains readily with all the ordinary stains, but Place especially recommends methylviolet, while Barker recommends a weak solution—25 per cent—of carbofuchsin. It is anaerobic, nonmotile—according to most observers, Gram negative, and does not form spores. Miss Ruth Tunnicliff isolated organisms from cases of Vincent's angina on slants of ascites agar, and concluded that strains of fusiform bacilli isolated in pure culture from the normal mouth, ulceromembranous angina, gingivitis and noma, appear culturally and morphologically the same organism. Cultures show that bacilli and spirilla are different forms of one organism. But whether spirilla formed from fusiform bacilli are the same as those found in the lesions themselves cannot

be decided, because—as Reckford and Baker point out—no one has ever been able to reproduce the lesions in experimental animals.

2. The spirochetes which invariably accompany the fusiform bacilli—whether a separate organism, as has been positively asserted by Krumwiede and Pratt, or only different stages in the life history of one organism, in accordance with Miss Tunnicliff's observations—are slender, of fairly large size, pointed at the ends, Gram negative and stain only feebly with ordinary dyes, though much more readily than does the spirocheta pallida. They are thicker than this latter organism also, appearing yellowish rather than white, with no geometric regularity to the spirals. They show on an average from five to eight wide irregular convolutions.

#### CLINICAL PICTURE.

Even though Vincent's conception of its etiology may now be in a measure superseded, his original clinical description still stands unrevised, and may be rendered into English about as follows:

In the greater part of the observed cases, this diphtheritic angina is located upon one of the tonsils, very rarely upon both of them. Usually it simultaneously involves one tonsil and its adjacent pillar. At the beginning of the infection the tonsil is covered by a whitish or grayish plaque, not very thick, soft in consistency, and easily detached by scraping. It covers an eroded and easily bleeding surface. The edges of this membrane are often very irregular, gradually extending until they cover the entire tonsil. When this has occurred it will be found much more difficult to detach the membrane, and if this is done it will be found to have formed again when inspected on the succeeding day. In mild cases the membrane will be small and incomplete after once being removed, but if the infection is severe, the diphtheritic membrane will be seen to cover a veritable ulcer, anfractuous yet superficial, caused by necrosis of the tonsillar tissue.

About the third or fourth day this pseudomembrane becomes thick, soft and almost caseous upon its surface, imparting a foul odor to the breath. It is now more easily detached than at the onset. The mucous membrane surrounding the ulcerated

area is edematous and erythematous, and the patient complains that the pharynx is very dry and swallowing difficult.

In mild cases the submaxillary glands will be but slightly enlarged, but this is not the case when the infection is of greater extent or the angina has not received any antiseptic treatment. In none of my cases did this adenitis break down into suppuration.

The angina is accompanied by pains in the back and loins, loss of appetite, catarrh of the upper respiratory tract, and rise of temperature, often slight, but nevertheless constant. Frequently, in the first few days of the attack, the temperature will register 38.5 or even 39 (degrees centigrade), but after the application of local treatment it will quickly fall to the neighborhood of normal, often by the third day, when the diphtheritic exudate has not yet been eliminated.

Usually the tonsil will be clear by the eighth or tenth day, and when the debris of the pseudomembrane which is still retained in the crevices of the ulcer have been eliminated, recovery is rapid. Occasionally we may encounter a case so stubborn that it will endure a fortnight or even longer.

In a later publication Vincent differentiated two clinical forms, which have been thus summarized by Amsden:

1. The ulceromembranous, by far the more common, in which both the bacillus and spirillum are found. The initial symptoms are those of an acute tonsillitis or pharyngitis, malaise, headache, fever—generally slight—and dysphagia. Objectively, there is at first redness of fauces and tonsils, fetid breath, coated tongue and swelling of the submaxillary glands. Later, on tonsils and pillars is seen a soft, friable, yellowish slough, slightly adherent, with marked ulceration of subjacent mucous membrane, easily bleeding on touch. Deep and extensive ulceration of tonsils, pillars and palate may follow, with loss of tissue and subsequent contraction.

2. The diphtheroid variety is very rare, about 2 per cent of all cases. It is due to the fusiform bacillus alone. The distinguishing feature of this form is a distinct false membrane, resting on an inflamed, slightly ulcerating base; clinically the two forms are otherwise alike. Rolleston claims that there are not two distinct varieties of the disease, the ulcerative being only the later stage of the membranous. Cocks recognizes two

types, the tonsillar or mild type, and the severe form, in which the membrane extends to extratonsillar structures, with ulceration, and accompanied by more severe general symptoms.

Sutter calls attention to the "curious fact" that rapid recurrence takes place when the membrane is removed, so similar to the membrane of diphtheria. In a number of cases reported the tonsils have been amputated, but, in each case, the wound became infected with the same organisms. The submaxillary, retromaxillary and cervical lymph nodes are often markedly swollen and tender, but rarely suppurate. The disease may be acute or subacute and often becomes chronic, the ulcers persisting for weeks or months. One attack is likely to be followed, months or even years later, by a recurrence. Pereira states that the "shortest duration of Vincent's angina in the adult is three weeks. In children it is usually shorter, for in them we do not see, as is the case with adults, cases of angina so prolonged as to become chronic."

#### DIFFERENTIAL DIAGNOSIS.

While, as Theisen has said, it is now an accepted fact that the finding of the fusiform bacillus with the spirillum or spirochete, in smears from throat swabs, makes the diagnosis of Vincent's angina easy and positive, it must be remembered that this bacillus is not the specific organism of Vincent's angina only, but is found also in cases of mastoiditis, bronchopneumonia, diphtheria, hospital gangrene, throat syphilis and stomatitis. Cultures and smears usually show mixed infections with other organisms, such as the Klebs-Loeffler, pneumococcus, streptococcus and staphylococcus. Mistakes in diagnosis are frequently made because cultures only were taken, and not throat swabs. Microscopic examination of smears from throat swabs will easily confirm the diagnosis. This author is firm in his belief that bad teeth, with the attending spongy condition of the gums, are among the most important etiologic conditions in Vincent's angina; Reckord and Baker think it probable that "the considerable use of candy or proteins with the subsequent lack of oral cleanliness predisposes markedly to this affection"; likewise, that poor teeth harbor the organism and that it will manifest itself in its characteristic lesions whenever a suitable opportunity is given.

In uncomplicated cases Carroll believes that we can often make a diagnosis and "put our minds at rest whilst awaiting laboratory corroboration." The exudative membrane, seen at the first examination, has a pearly white color, with abrupt thickened edges, and can be readily taken off, when the underlying mucous membrane will seem to be of practically normal appearance, though in the ulcerative type erosions or ulcerations will appear underneath. The surrounding tissue, differing greatly from most other throat inflammations, will look about as usual, no involvement being evident. In diphtheria, the membrane is grayish or dirty gray in appearance, is very tenacious, and, if removed, usually tears or breaks, leaving behind many minute bleeding points. The contour of the patch will be irregular in outline and the surrounding tissues red and angry looking.

Streptococcic sore throat presents a membrane, if coalescence of the exudation points has taken place, which closely resembles the slough seen in Vincent's disease, but the surrounding tissues will be red and "raw looking," and general constitutional involvement much more in evidence—high temperature, rapid pulse and, in the worst cases, marked prostration.

Syphilis, while often confused with Vincent's angina, should not present any difficulties when the proper technic is employed. From primary syphilis, the absence of infiltration and of any marked adenopathy, and its rapid clearing up under local treatment, should serve as sufficient differential points. Fusiform bacilli and spirochetes are found in syphilitic ulcers, but the ulcer has none of the clinical features of true Vincent's angina. The Wassermann blood test will also help in making the differentiation. It is possible, however, for Vincent's angina to be superimposed upon an oral or buccal syphilitic infection. Concerning this possibility, Barker and Miller have said:

"Throughout the literature scattered contributions are found in which it is maintained that in uncomplicated cases of 'Vincent's angina' the Wassermann reaction is often found positive. This view is held by St. Clair Thompson, Much, Sobenheim and others. Critical analysis of the cases thus reported shows that this statement rests on decidedly flimsy evidence.

Those who have had the greatest experience agree that though a positive Wassermann does not exclude Vincent's disease, and though in some isolated cases neither the clinical appearances nor the microscopic findings are sufficient to establish a definite diagnosis with certainty, none the less serologic findings are invariably negative in uncomplicated cases of Vincent's disease. Recently Taylor and McKinstry have conducted studies to clinch this point. From a large number of cases of Vincent's disease, fifty-five were chosen at random; fifty-three gave absolutely negative Wassermann reactions, while two were positive. Both of these patients admitted a primary infection. The authors conclude that 'the prevailing belief in the occurrence of a positive Wassermann in Vincent's angina has no foundation in fact, and that the two conditions can be differentiated with absolute certainty by the application of bacteriologic and serologic methods. When a Wassermann reaction is positive in cases of Vincent's angina, then a double infection exists, either as a coincident syphilitic and Vincent's infection, or as the occurrence of Vincent's angina in a subject with latent syphilis.'

#### TREATMENT.

As in many other conditions, prevention is the best treatment. Lack of oral cleanliness is probably the chief reason for the existence of Vincent's angina, and when proper mouth hygiene becomes universal it will wholly disappear. Until this millennial condition shall be approximated, we must resort to local measures.

Examination of the literature shows that a wide range of therapeutic agents has been employed at different times, with many claims of completely successful results. Place, in 1911, found "the most satisfactory of the local applications, and one universally successful in a few days," to be "swabbing with hydrogen peroxid full strength or diluted one-half, until the ulcer is pretty clean, and then painting with 2 per cent solution of chromic acid, once daily. The peroxid is used for its free oxygen and its mechanical effect in removing membrane so that the chromic acid may reach the base of the ulcer. The combination worked better than either alone. Cure occurred rapidly in from two to six days, with hardly an exception."

Emrys-Roberts recommended a lotion consisting of hydrogen peroxid, 5 fluid ounces; ipecacuanha wine, 3 drams; glycerin, 5 drams; and water enough to make eight ounces. The rationale of this formula, the originator tells us, is based on the following considerations: First, that hydrogen peroxid is especially useful in loosening and cleaning up purulent exudate, and moreover would militate against the growth and multiplication of the anaerobic fusiform bacillus; secondly, that ipecacuanha wine might prove to have a more or less specific action upon the spirochetes, and thirdly, that glycerin, by virtue of its hygroscopic and penetrating properties, would effectually convey the preceding ingredients into otherwise inaccessible recesses.

Barker and Miller suggest that liquor potassii arsenitis (Fowler's solution) be added to this formula. "The ulcers are swabbed with this solution two or three times a day, and all patients, whether the gums are affected or not, are instructed to put ten to fifteen drops on a toothbrush, twice daily, and to brush the teeth and gums vigorously. Improvement and cure are rapid."

Other local measures recommended are the application of tincture of iodine, colloidal silver, flavin and powdered methylen blue. The best local application is undoubtedly arsphenamin, dissolved in glycerin. The parts to be treated are thoroughly cleansed and dried, the arsphenamin solution applied with a small cotton swab, and the substance rubbed into the lesions.

Far better, however, is the intravenous injections of arsphenamin, following the regular antiluetic technic. Quoting from Davis and Pilot: "On the basis of the marked results in spirochetal diseases, such as syphilis and yaws, arsphenamin has been extended in its use, to other conditions, particularly infections in which the Vincent spirochete plays a rôle. Encouraging results have been obtained in the local and intravenous introduction of arsphenamin in Vincent's angina and gingivitis. It should be remembered that while the arsphenamin has spirocheticidal action, it may not directly influence the associated organisms—i. e., the fusiform bacillus and the pyogens. The best results may be expected in lesions which reveal the spirochetes as the predominant infecting organism."

Numerous reports of the efficacy of this treatment are to be found in the more recent literature on Vincent's angina. Späno, an Italian pediatrician, employed it with signal success in the treatment of little children, giving three or four injections at three day intervals, in doses ranging from 5 to 10 cg., according to the age of the child. Sebenq, in his Paris thesis, testifies to "the remarkable therapeutic action of arsenobenzol, which constitutes the treatment of choice." Pareira devotes a large part of his thesis, published in 1921, to a consideration of the arsphenamin therapy, which was first introduced by his master, Achard, and after detailing a number of cases where both local and intravenous treatment was given, states in his conclusions that "If the treatment of choice in Vincent's angina in its common form is the local application of salvarsan or neosalvarsan, in the more serious and extensive forms of the disease local application should give place to intravenous injection, which, in diminished dosage, at sufficiently long intervals, appears to exercise a specific action upon this malady."

A more recent report from France—November of last year—by Sargnon and Trossat, testifies to the rapid amelioration of all the symptoms of a puzzling case of ulceromembranous laryngitis, when intravenous injections of neosalvarsan were given. Similar testimony can now be found in many publications made recently in this country.

My own opinion and experience as to the value of arsphenamin therapy in Vincent's angina can best be recorded by citing the history of various patients who have come under my care.

#### CASE REPORTS.

Case 1.—On May 16, 1921, I was summoned to see a young lady who had been in bed for two or three days, the temperature ranging around 103°. Examination showed the tonsils and uvula covered with a leathery yet macerated membrane, giving off a foul odor. A diagnosis of diphtheria was made, and antitoxin was at once administered. When the patient had received 17,000 units from a 20,000 unit tube, she volunteered the information that five years before she had received a similar administration of antitoxin for supposed diphtheria. The culture taken for examination proved negative for diphtheria, and the day following the administration of the antitoxin there



was no change in the appearance of the membrane, and no improvement in the general systemic condition. A smear, which was now examined microscopically, gave unmistakable testimony to the presence of Vincent's organisms, and following the intravenous injection of salvarsan, combined with local application of iodine, there was immediate improvement, soon followed by complete cure, so that in three weeks' time I was able to remove the tonsils under local anesthesia.

Case 2.—A music teacher, 26 years old, had been ill a week with an intensely painful throat affection, which was thought to be tonsillitis. She was hardly able to take any nourishment and had been quite unable to continue giving singing lessons. When I inspected the throat, on November 30, 1923, the tonsils were large and "boggy," and, together with the uvula, showed punched out ulcers covered with a membrane. The breath was very foul, the pulse rapid and the temperature registered 101. A full dose of salvarsan was at once injected, while codein and aspirin were given to allay the pain and fever. The throat condition improved following the first injection; a second was administered three days later, and although convalescence was rather more protracted than usual, the patient was well in three weeks' time.

Case 3.—This patient, an express messenger, aged 19, had had scarlet fever four years ago, and for many years had had annual attacks of "acute tonsillitis." When I saw him on January 1 of this year, he had been suffering from a severe sore throat for ten days. I treated him and had planned to remove his tonsils, but found an acute albuminuria, so that I advised that tonsillectomy be postponed until after the kidneys had cleared up. He went home to another city. On March 7 he again called to see me with a temperature of 104 and a severe sore throat. He said that he had been seen by several doctors and a dentist, none of whom had done more than postulate "bad tonsils" and "sore throat." There were large punched out ulcers upon both tonsils and upon the pharynx as well. The glands of the neck showed definite enlargement as the patient was suffering extreme pain. Examination of smears from throat and mouth and gums disclosed characteristic Vincent's spirillum, the tonsils, pharynx and gums all being involved in the infection. A full initial dose of salvarsan was

given intravenously with local application of sodium perborate. The pain was relieved after first injection and a second was given in five days, which completed the cure within a fortnight.

Case 4.—A commission merchant, aged 37, presented himself, with the cervical glands enlarged and painful, and a history of great pain and difficulty in swallowing. Inspection showed punched out ulcers with a membranous covering on each large "boggy" tonsil, which gave off a foul odor. A smear made from the throat showed the Vincent spirillum, so a diagnosis of Vincent's angina was immediately made, and marked relief obtained within twenty-four hours by the local application of 5 per cent mercurochrome and intravenous injections of salvarsan. The throat was completely normal within a week's time.

Case 5.—Mrs. N., who consulted me on February 20, 1924, gave a history of intermittent sore throat for the past two years. Several different physicians had treated her with gargles and local applications, all of which had failed to effect any permanent benefit. Inspection showed ulcers upon the tonsils and pharynx, and a smear taken from a throat swab returned a positive report of the presence of Vincent's organism. Salvarsan was immediately injected, with local applications of sodium perborate, and improvement was so immediate as to be almost spectacular. Within three days the ulcers were practically healed and the patient was able to eat in comfort, which she said she had not been able to do for many months previously. As this patient was 61 years of age, and the condition had been unrelieved for so long a period, the results of the salvarsan treatment seem especially brilliant in this case.

#### REFERENCES.

- Amsden, H. H.: A Case of Vincent's Angina Treated With Neosalvarsan. *Med. Rec.*, May 9, 1914.  
Amsden, H. H.: Vincent's Angina. *Med. Rec.*, November 21, 1914.  
Barker, L. F., and Miller, S. R.: Perforating Ulcer of the Hard Palate Resembling Tertiary Syphilis, But Due to a Fusispirillary Invasion (so-called Vincent's Angina). *J. A. M. A.*, 71:193, Sept. 7, 1918.  
Carroll, J. D.: Vincent's Angina. *Albany M. Ann.*, 43:72, 1922.  
Cocks, G. H.: Vincent's Angina. *Laryngoscope*, September, 1913.  
Davis, D. J., and Pilot, I.: Studies of Bacillus Fusiformis and Vincent's Spirochetes. *J. A. M. A.*, 79:944, Sept. 15, 1922.

- De Sanctis, A. G.: Vincent's Angina. N. Y. Med. J., November 6, 1915.
- Emrys-Roberts, E.: A Note on the Treatment of Vincent's Angina. Brit. M. J., 2:360, 1917.
- Graves, Stuart: Acute Vincent's Angina Yielding Only to Treatment With Salvarsan. Kentucky M. J., 20:445, July, 1922.
- Halsted, T. H.: Vincent's Angina: Its Frequency and the Importance of Its Recognition. Trans. Am. Laryngol. Assn., p. 216, 1912.
- Krumwide and Pratt: Fusiform Bacilli: Cultural Characteristics. J. Infectious Diseases, 13:439, 1913.
- Pereira, A. M.: L'angine de Vincent a marche extensive et son traitement par l'injection intraveineuse de neosalvarsan. These de Paris, No. 432, 1921.
- Place, E. H.: Vincent's Angina. Boston M. and S. J., 165:720, November 9, 1911.
- Reckord, F. F. D., and Baker, M. C.: Vincent's Angina Infection. J. A. M. A., 75:1620, December 11, 1920.
- Richardson, C. W.: Vincent's Angina. Ann. Otol., Rhinol., and Laryngol., March, 1909.
- Sargnon, A., and Trossat, H.: Case of Primary Ulceromembranous Laryngitis of the Vincent Type. Oto-rhino-laryngol. Internat., 7:633, November, 1923.
- Sebenq, Charles: L'angine de Vincent. These de Paris, No. 139, 1919.
- Spaño, R.: Neoarsphenamin in Ulcerative Stomatitis. *Pediatrics* (Naples), 30:241, March 15, 1922. Abstract in J. A. M. A., 78:1426, 1922.
- Sutter, C. C.: Ulceromembranous Angina (Vincent's) and Its Treatment; With Report of Cases. Med. Rec., March 8, 1913.
- Taylor, F. E., and McKinstry, W. H.: A Serilogic Investigation of Vincent's Angina. British M. J., 1:82, 1918.
- Theisen, C. F.: The Clinical Course and Treatment of Vincent's Angina. Albany M. Ann., 40:224, 1919.
- Vincent, H.: Sur l'etiologie et sur les lesions anatomopathologiques de la pourriture d'hôpital. Ann. de l'Institut Pasteur, 10:488, 1896.
- Vincent, H.: Sur une forme particuliere d'angine diphteroïde (Angine à bacilles fusiformes). Bull. et mem., Soc. méd. des hôp. de Paris, 15:244, 1898.
- Vincent, H.: De la frequence de l'angine à spirilles et bacilles fusiformes. Presse Méd., March 29, 1905.

DEFLECTED SEPTUM IN A PATIENT IN WHOM  
A FATAL RESULT FOLLOWED.

BY GEORGE H. THOMPSON, M. D.,

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I. B., age 25, came January 2, 1926, stating that he could not breathe properly through his nose. Examination showed the septum thickened, with a bowing opposite the middle turbinate on the left side and a strong bony spur formation on the left floor. Advised to have a submucous resection done and reported for it a month afterward. His past history was without interest, except that between the ages of 20 and 22 he was supposed to have pulmonary tuberculosis and took a cure for it; since that time he had been in robust health. The only thing noticed at the time of the operation was that he seemed excessively nervous. I remarked on this at the time because when I had examined him one month previously he had not struck me as being a nervous fellow. The operation was without accident of any kind, but the rest of the day he oozed a great deal more than these patients usually do, and during the night was sick at his stomach. However, his temperature and pulse were normal both during that day and until 3 o'clock the afternoon of the next day, when his temperature was noted to be 102.6 and pulse 120. At that time his physical examination was negative and the only complaint that he had was he felt nervous. Sedatives were administered and cold sponge baths ordered, but at 12 o'clock that night his temperature was 106 and his pulse was rapid and weak. Large doses of tincture of digitalis, by the bowel, and adrenalin, hypodermically every two hours, and saline intravenously caused his pulse to improve, and his temperature dropped to 104 and he had a fairly comfortable night. The following morning, however, his temperature was again 106 and his pulse was rapid and weak, and anything that we could do for him was without avail. His body,

from his neck to his pelvis, was the color of a broiled live lobster. His nervous system, so far as physical signs went, was negative. The spinal fluid was clear, came away without pressure, and was normal both as to cell count and chemical reaction. He had no headache at any time and apparently had little or no resistance to the infection, as when his temperature was 106 his leucocyte count was only 6,500. He died 26 hours after the onset of his acute illness. The medical consultant, who saw him about four hours before his death, found he had a consolidation of the right upper lobe, and his diagnosis was streptococcus pneumonia.

There was no blood in his urine, so I suppose the blush on his body was not due to the breaking down of the red corpuscles. What part, if any, the operation played in this man's death is, of course, left to conjecture. One thing is quite sure, all that we were able to do for him had no effect on the progress of the disease.

PARANASAL SINUS DISEASE AND ASTHMA IN  
YOUNG CHILDREN.\*

BY DEAN M. LIERLE, M. S., M. D.,

IOWA CITY.

During the last eighteen months the department of pediatrics referred twenty cases of asthma to the department of head specialties for examination and treatment. One case of five years' duration has been added to this list. The diagnosis in every case has been made by the department of pediatrics. We wish to emphasize the fact that we are not considering paranasal sinus disease as an etiologic factor, but we are presenting a few interesting facts concerning the history, various laboratory tests and the treatment of the sinus condition.

The youngest child was two years of age and the oldest was fifteen. Eight were girls and twelve were boys. A family history of asthma was present in forty per cent of the cases. In many cases the asthmatic attack was preceded by a chronic bronchitis and in many during the prespastic stage described by Haseltine various symptoms were manifest, such as urticaria, eczema, eosinophilia, cyclic vomiting, arthritis, and frequent attacks of tonsillitis. Several cases who were sent primarily for the treatment of eczema have had attacks of bronchospasm in our ward.

In nineteen cases a "head cold" immediately preceded the bronchospasm and one denied any relationship to "head cold" but admitted the attacks were more frequent and longer in duration during the winter months. In practically all the asthma was worse in the fall, winter and spring, and especially during the wet rainy season. Two reported the attacks were as frequent and severe in the summer as at any other time.

Foreign protein tests were done both in the departments of pediatrics and head specialties. All but three had negative reactions to the tests. Two were sensitive to chicken feathers,

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\*Department of Head Specialties, University of Iowa.

but an attack could not be produced when in their presence. One was sensitive to corn and a typical bronchospasm could be produced by the ingestion of corn. The last patient, however, also had attacks of asthma when his diet was free from corn and he was markedly improved by the treatment of the paranasal sinus disease.

Dr. Jane Adam of Glasgow, in the June, 1925, number of *American Medicine*, writes: "Week end asthma, i. e., asthma on Sunday or Monday, is common in school children and wage earners who have an easy time and a big feed at the week end." Two of our cases reported having had asthma following the week end. However, the parents said that at this time they also had "head colds."

A few interesting points were observed regarding the blood and urine. Polyuria was present in about fifty per cent of the cases during attacks. In a large number indican was present in a conspicuously large amount and the urine was highly alkaline. Some writers have found that the blood calcium was low in asthmatics. Kempinski says that calcium in many cases exerts a great influence on the vegetative and autonomic nervous systems. With this in view the blood chemistry was done in twelve cases. The blood calcium was to the normal limit in every case, ranging from 10.4 milligrams to 100 c. c. to 11.5 milligrams. The P. H. ranged from 7.30 to 7.38. Chlorides, as sodium chloride, ranged from 0.475 to 0.531.

Upon examination, paranasal sinus disease was present in every case, and it was found that an ethmoiditis of varying degrees was also present. In nine, a chronic suppurative pansinusitis was present. A marked hyperplastic ethmoiditis and sphenoiditis was present in six cases. In three, a chronic suppurative ethmoiditis bilateralis was found, and in four, a chronic suppurative maxillary sinusitis was present. The faucial and pharyngeal tonsils were present and chronically infected in ten children. In one case the lingual tonsil was so large and badly infected it was necessary to remove it by electric cautery. In sixty per cent spurs and deflections of the septum, causing obstruction to sinus drainage, were found.

The treatment of paranasal sinus disease is long and tedious and hospitalization is necessary. We wish to emphasize the fact that as long as the patients are in the head specialties

service they are also under the personal supervision of the pediatricist who makes daily rounds on the ward. One of the most important points of the treatment is the diet.

Dr. Amy Daniels, of the Children's Hospital, has shown that children with paranasal sinus disease do well with a diet high in fat soluble A vitamin. She has observed that rats fed diets adequate in all other respects but low in vitamin A factor in the course of time develop infections of the upper respiratory tract. Such animals manifest many of the symptoms of human beings suffering from similar types of infection; for example, loss of appetite, failure to gain (in children), gastrointestinal disturbances, snuffles, paranasal sinus disease and death unless appropriate treatment is introduced. Macroscopic examination of the various organs of such animals, with the exception of the nasal sinuses which have been found filled with pus, shows nothing consistently abnormal. Occasionally, the lungs are infected; often the intestines are filled with gas and are greatly distended. Death in these animals seems to be due to an infection superimposed on tissues which have been altered by the dietary deficiency. In those cases where the animals have been able to continue for long periods of time on the restricted rations peculiar eye conditions characteristic of xerophthalmia have developed.

Investigations by Blegvad ("Vitamin a Deficiency in Denmark and its Results," *The Lancet* in Volume CCVI, 1206-1924) on the incidence of xerophthalmia in Denmark has shown that the disease is inversely proportional to the consumption of butter fat (milk, butter and cheese) which is the chief source of vitamin A in the diet of the Nordic race. During the earlier years of the war, when dairy products were being more largely exported, there was a considerable increase in the number of cases of xerophthalmia.

Dr. Daniels has found that in certain sections of the United States, where sinus infection is prevalent, the consumption of butter fat is low. For example, one would expect that the consumption of butter fat in Iowa would be great, but she has found in an extensive study of Iowa communities that this is not true. In accordance with Dr. Daniels' observations the patients are given foods high in fat soluble A vita-



min, such as butter, milk, eggs, carrots, spinach, lettuce and tomatoes. In addition to these, cod liver oil is given in every case. It is very important that the feeding be supervised, for the child as a rule will eat only the foods of his liking.

The routine operative procedure is as follows: The patient is put to sleep under ethylene anesthesia and a nasoscopic examination is done. If the X-ray has shown a blurred maxillary sinus, the sinus is aspirated, and if macroscopic pus is found in the antrum, a fairly large opening is made in the inferior meatus without interfering with the anterior end of the inferior turbinate. Following this, if present, the faucial tonsils and adenoids are removed. If on nasoscopic examination a suppurative ethmoiditis and sphenoiditis is present, operative work in this region is avoided except in cases where all other procedures have failed. In only one case in this group has the evisceration of the ethmoids and sphenoids been necessary. As a rule, the ethmoiditis and sphenoiditis will respond to conservative treatment after all other foci have been removed. In this same case it was also necessary to do a Caldwell-Luc operation to clear up the maxillary sinus infection. The antrum in this instance contained pus and a remarkably thickened polypoid membrane. High septal deflections were not corrected on account of the danger of nasal deformity. However, in several cases, low septal spurs which interfered with the drainage of the maxillary sinuses were removed.

The postoperative treatment is simple, yet very important. On the second day following the operation, daily nasal irrigation, followed by the dropping of five per cent argyrol into each side of the nose, is instituted. If an opening has been made in the maxillary sinuses they are irrigated on the fifth day with a warm boric solution followed by five per cent argyrol or two per cent mercurochrome. In many of the younger children it is often necessary to treat the sinuses under ethylene anesthesia by Dr. Dean's method. As a rule, the sinuses respond readily to treatment and the patients are discharged to return at regular intervals for reexamination.

In a large majority of the cases the results have been very gratifying. Two have reported that they have been free from asthma for one year. Seventeen have reported marked im-

provement, five of whom have had only two slight attacks within a year.

I wish to report one case which is rather typical of the entire group. R. A., aged twelve, was transferred from the medical service for the removal of the foci of infection. Their diagnosis was bronchial asthma. All foreign protein tests were negative. For as long as the patient could remember he has had long and frequent attacks of asthma which were present throughout the year but were worse during the winter, spring and fall. They were always preceded by a "head cold" and with profuse anterior and posterior discharge. He denied paranasal sinus pain but admitted frequent attacks of acute tonsillitis and complained of cough and sputum especially after the asthma had begun. On examination he was found to have a hyperplastic ethmoiditis and sphenoiditis with polyps bilateral; chronic suppurative maxillary sinusitis bilateral, worse left; deflected septum; chronic faucial tonsillitis and adenoids and cervical adenitis. His tonsils and adenoids were removed, but no improvement was noted. Following this a Dencker operation was done on the left maxillary sinus. Very little improvement was noted, and it was necessary to do a Sluder operation on the ethmoids and sphenoid on each side. This was followed by a marked improvement, and only one attack of asthma was reported in a period of six months. On re-examination infected granulation tissue was found in each ethmoid region, and pus was present in the left maxillary sinus. Each ethmoid region was recuretted and a Caldwell-Luc operation, left, was done. He was discharged and the parents have reported that he has been free from asthma for the last nine months.

XXXIII.

PRESENTATION OF CASE OF BRONCHIECTASIS  
IN A CHILD ASSOCIATED WITH PARANASAL  
SINUS DISEASE.\*

By A. J. CONE, M. D.

IOWA CITY, IOWA.

Case.—L. I., 10 years old, admitted to the State Sanatorium for Tuberculosis at Oakdale, October 9, 1925. She was referred to the Head Specialty Service, University Hospital, for study.

Present illness: The patient had influenza last summer. This was followed by a relapse, after which the mother noticed that the patient coughed and raised a great deal of sputum. In September the patient reported pain along the right costal margin. The diagnosis made at that time, after consultation, was pulmonary abscess. A laboratory examination showed acid fast bacilli. Apparently only one smear was submitted.

Past medical: The patient had measles at two, with good recovery. At four years of age the patient had an attack of chills and fever which was followed by pain and swelling in the chest to the left of the spine. This was opened and tube drainage was necessary for two months. The patient has coughed a little ever since that time. The coughing was always more severe during colds or other infections. The patient had influenza in 1918. Four years ago the patient had tonsils and adenoids removed under anesthesia with good recovery. Family history negative.

Patient was admitted to Oakdale, October 9, 1925. All findings were negative for tuberculosis. Their impression was nontuberculous lung infection, with question as to bronchiectasis, or empyema with bronchial fistula.

Patient was transferred to Children's Hospital, Pediatric Department, November 3, 1925. Repeated sputum examina-

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\*Presented at the fourth annual meeting of the Dean Clinical Society.

tions for tuberculosis were negative. Findings showed evidence of a chronic bronchiectasis with etiology in upper respiratory tract.

Patient was seen on requisition by the Department of Head Specialties.

The following diagnosis was made:

1. Paranasal sinus disease.
2. Thickened septum.
3. Diseased lymphoid tissue in naso- and oropharynx.

X-ray at this time showed blurring of the maxillary sinuses; no frontals. Patient was transferred to the Department of Head Specialties for eradication of foci in upper respiratory passage and treatment of bronchiectasis. The only additional note on admission was a history of frontal headache.

Treatment.—Examination of sinuses: Removal of faucial tonsil stumps, and diseased lymphoid tissue in nasopharynx, under chloroform ether anesthesia.

Nasoscopic examination: On the left side much muco-pus was found filling nares, apparently coming from ethmoid and sphenoid regions. On the right side there was pus in the middle meatus and sphenoid fissure. On aspirating, pus was obtained from each maxillary sinus. Meatal openings were made under each inferior turbinate. Very small tags of lymphoid tissue were removed from each tonsillar fossa and nasopharynx.

Laboratory report on washings: Right antrum showed pus cells and organisms; short chain streptococci, diplococci, and short rods. The left antrum showed large numbers of pus cells, but no organisms. The postoperative period was stormy with temperature at 102° and pain in chest. Following the operation and initial sinus examination, the patient's antra were irrigated in clinic without anesthesia, but because of resistance to local treatment, the irrigation of the antra and treatment of the ethmoid and sphenoid regions was done under ethylene anesthesia by Dr. Dean's method. On the 15th day of December, 1925, under chloroform anesthesia, the first bronchoscopic study was made. Right bronchus was found dilated and contained much pus. Left was not apparently dilated and contained comparatively little pus.

On December 21, 1925, under chloroform anesthesia, lung mapping was done with lipoidal injection. X-ray plates showed dilatation at right base posterior.

Diagnosis: Bronchiectic cavity at right base posterior. From this date on bronchoscopic treatment was given under chloroform anesthesia; cleaning out dilated area with suction followed by injection of lipoid. At the same time the sinuses were treated.

The above procedure was repeated at seven to ten day intervals. Between times the patient received postural treatment; daily nasal irrigation, argyrol drops in nose, and special sinus diet.

There has been a noticeable decrease in the amount of sputum, the nasal condition has improved, the patient is stronger and much more active and is gaining in weight.

With the exception of treatment, this case follows closely the literature on association of sinus disease and bronchiectasis. W. W. Mullin, in his article "The Accessory Sinuses as an Etiologic Factor in Bronchiectasis," states that patients with bronchiectasis will usually be found to have a well marked sinusitis. In his experimental work he has demonstrated the lymph drainage of the antrum is by way of the submaxillary and deep cervical nodes. From these nodes drainage passes onward by the cervical lymph ducts, thence into the great veins, the right side of the heart and the pulmonary artery to the lungs. The frontals drain by the same route. He also mentions pulmonary involvement by inhalation. Another point is that bronchiectasis does not occur in the majority of cases of chronic bronchitis and the tendency is to get well unless fostered and fed by chronic sinus infection.

Mitchell and Shea's article on "Nasal Sinus Disease in Children" states that influenza appears to have a predilection for infecting the sinuses generally, and the present increase in the number of sinus diseases may be attributed to the recent epidemic. The authors also mention the importance of diet from the etiologic as well as the treatment side of the question.

In Dennis' article on "Accessory Nasal Sinus Infection with Complications," twenty-four patients in his group of cases had bronchiectasis. Ten cases dated trouble from an attack of influenza. Tuberculosis had been the most frequent mistake

in diagnosis. In his summary he states that infection in the sinuses, particularly in the maxillary, is so commonly found in bronchiectasis as to strongly indicate an etiologic relation.

Webb and Gilbert report the most frequent sinus condition found associated with bronchiectasis and bronchitis as a bilateral empyema of the antrum.

It is Dr. Dean's opinion that the chronic paranasal sinus suppuration, by infecting the bronchi by inhalation route, interferes with the healing of the chronic lung suppuration and the sinus disease should be treated at the same time the bronchoscopic treatment is given. His view is based upon the bronchoscopic findings of blood in the bronchi after operations on the nose and throat under a general anesthesia, even with the head lowered; the same factors entering as in the course of pus from sinuses to the lungs during sleep.

#### BIBLIOGRAPHY.

1. Mullin: *Annals of Otology, Rhinology and Laryngology*; 30: 683; September, 1921.
2. Dennis: *Annals of Otology, Rhinology and Laryngology*; 33: 451; June, 1924.
3. Mitchell and Shea: *Archives Pediatrics*; 39:230; April, 1922.
4. Webb and Gilbert: *Journal American Medical Association*; 76: 714; March, 1921.

XXXIV.

A STUDY OF THE NOSE AND THROAT IN EIGHT  
CASES OF DIABETES IN CHILDREN.\*

By T. S. BURGESS, M. D.,

IOWA CITY, IOWA.

On January 21, 1926, eight cases of diabetes mellitus were referred in a group to the Head Specialty Service of the University Hospital from the Department of Pediatrics, for nose and throat examination for foci of infection. All sinuses were X-rayed and the plates read by Dr. Allen. All cases showing evidence of sinus disease on X-ray were examined nasoscopically, and the sinuses aspirated under the personal direction of Dr. Dean. The sinus washings were cultured and a full bacteriologic report made.

Case 1.—L. G., age 5: Has had several previous admissions, sugar being first found in the urine last September following an attack of measles.

Tonsils and adenoids were large and diseased; cervical glands were enlarged; and there was mucopus in each nares.

X-ray examination showed definite blurring of the right antrum, while the left was slightly blurred.

The nasoscope showed a little pus in the middle meatus of each side.

On aspiration of the right side pus so thick that it could be aspirated only with difficulty was obtained. From the left side pus was also obtained, but less than from the right, and it contained more mucus.

Case 2.—D. D., age 9: The classical symptoms of diabetes were noted about Christmas, 1925, and sugar was found in the urine.

Tonsils and adenoids were large and diseased; cervical glands were enlarged; and there was no pus in the nares.

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\*Presented at the fourth annual meeting of the Dean Clinical Society.

X-ray examination of the sinuses was negative.

Case 3.—B. S., age 12: Is a case who has had diabetes for some time, and had many previous admissions for diet adjustment.

She had had a tonsillectomy three years previously; and tonsils and adenoids were out clean. There was slight cervical adenitis.

X-ray examination of the sinuses was negative.

Case 4.—B. W., age 6: Also has had diabetes for some time and has had several previous admissions for diet adjustment. Tonsils and adenoids were large and diseased; cervical glands palpable; there was no pus in the nares.

X-ray examination of the sinuses was negative.

Case 5.—B. D., age 5: Last Summer symptoms of diabetes were noted for the first time, but there was no sugar in the urine. In January there was a recurrence of symptoms, and sugar was found in the urine.

Tonsils and adenoids were large; cervical glands were enlarged, and there was mucopus in each nares.

X-ray examination showed definite blurring of the right and very slight blurring of the left antrum.

The nasoscope showed an abundance of pus in the middle and superior meati of the right side, while the left side was clean.

Washings from the right antrum contained thick white pus. Those from the left were clear.

Case 6.—C. H., age 12: Is a case of diabetes of some years' duration, who has had several previous admissions for diet adjustment.

Diseased tonsils and adenoids were removed in July, 1925. At present there is a little lymphoid tissue in the nasopharynx, and slight cervical adenitis, which is probably secondary to the sinus infection.

X-ray examination showed definite evidence of maxillary sinus suppuration, the right antrum being very markedly blurred and the left only slightly so.



The nasoscopic examination was unsatisfactory on account of the presence of so much mucus.

The antral washings from the left side were clear, while those from the right contained much thick pus.

Case 7.—G. F., age 12: Is also a case with several previous admissions for diet adjustment.

Tonsils and adenoids were large and diseased; cervical glands were palpable and there was no pus in the nares.

X-ray examination of the sinuses was negative.

Case 8.—R. S., age 2: Following an attack of what was diagnosed "intermittent influenza," sugar was first found in the urine last September.

Tonsils and adenoids were enlarged and diseased; cervical glands were palpable; and there was no pus in the nares.

X-ray examination showed blurring of both antra.

Patient developed acute mastoiditis and sinuses could not be examined further.

#### BACTERIOLOGIC REPORT.

B. D.—Right antrum. Smear: Gross heavy pus. Staphylococci in large numbers. Cultures: Staphylococcus aureus only could be isolated, though a smear from one culture contained one chain of small cocci.

Left antrum: Material consisted of clear watery fluid with flakes. No growth found in any of three separate cultures.

L. G.—Right antrum. Smear: Moderate mucopus with some blood. Cocci in pairs. One chain of cocci seen. Cultures: No growth obtained. Four cultures made.

Left antrum. Smear: Moderate mucopus. Cocci singly, in pairs, and in small groups. One chain of small cocci. Culture: Staphylococcus aureus only.

#### SUMMARY.

Eight cases of diabetes mellitus were routinely referred. Seven had diseased tonsils and adenoids, and one had had tonsils previously removed.

Four had blurred maxillary sinuses on examination. One had both definitely blurred; three had definite blurring of the right antrum and slight blurring of the left. Three had sinuses

aspirated. In every case where there was definite blurring on X-ray, pus was obtained.

Two with slight blurring of one sinus on X-ray had clear washings from the sinus, while from the third slightly blurred sinus thick pus was obtained.

Staphylococcus seemed to be the predominant organism. These investigations have been made too recently to determine any effect on the diabetic condition.

## XXXV.

### MEGAESOPHAGUS. REPORT OF CASE.\*

By O. D. CUNNINGHAM, M. D.,

IOWA CITY.

This patient entered the pediatric service of the University Hospital in November, 1924, at the age of 22 months, with the complaints of vomiting since birth, constipation and being unable to talk or walk. The vomiting was at times projectile, and the vomitus usually consisted of food eaten the day before, being always slimy and rancid, with the odor of butyric acid.

Fluoroscopic X-ray showed nothing suggestive of cardio-spasm. Plates at the time of the barium meal and five hours afterward showed barium in the intestine, and also above the stomach in a large dilation of the esophagus, and a threadlike opening into the stomach. The patient was given a course of atropin treatments with no benefit in the taking or retaining of food.

The general condition was fair for the very small amount of food taken, the general and mediastinal examination being negative except for some evidence of rickets and secondary anemia. The Wassermann was negative. The child was transferred to the Head Specialty Service, November 11, 1924, for study. Liquid bismuth was found to pass readily into the stomach, thicker porridge was retained in the dilation above the diaphragm.

Repeated attempts by Dr. Dean to see the outlet of the dilation by esophagoscopy and to pass a string or flexible bougie into the stomach were unsuccessful, because the blind end of the dilation was at a lower level than the outlet, and there was a kinking of the lower esophagus. A gastrostomy was advised.

On the first of February, Dr. Rowan operated and found the stomach much larger than normal. The triangular space between the crura of the diaphragm was wider than normal.

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\*Presented at the fourth annual meeting of the Dean Clinical Society.

The latter was thought to be due to the esophageal dilation and its contents pressing upon the diaphragm.

Following gastrostomy the child was fed by way of the fistula, and he became better nourished and stronger. However, repeated attempts to induce the child to swallow a string into the stomach were unsuccessful, as were also attempts to pass a string by retrograde esophagoscopy.

On March 31, 1925, the child was induced to swallow a thread weighted with a B B shot, camouflaged with soft candy. This passed into the stomach and was recovered through the gastric fistula. A heavier twisted silk cord was drawn through to replace it.

A No. 16 Tucker bougie was then passed readily without evidence of stricture; two weeks later, a No. 18, and three weeks after that, a No. 24. Then on alternate days Dr. Dean's No. 20 electrically heated bougie was passed and held in place for twenty minutes at a temperature of 42° C., followed by a No. 24 Tucker, so that by June the child was able to eat and retain soft foods. The gastrostomy foods were gradually omitted and the gastric fistula allowed to heal.

Due to irritative inflammatory lesions in the wall of the dilation, evidently from the presence of fermented foods earlier retained, the mucous membrane was treated from time to time with argyrol by esophagoscopy.

Because of dehydration, intercurrent infections and general debility, it was repeatedly necessary to revive the child from a very low state by intravenous and intraperitoneal fluids and by transfusion.

He was sent home in October, 1925, taking soft foods fairly well. The mother now brings the child back and says he has gained six pounds since his discharge from the hospital and has been looking better generally. He takes liquid and soft foods well, but regurgitates chunks. He is now three years of age, but is not yet able to walk or talk.

Megaesophagus, otherwise known as idiopathic dilation of the esophagus, and probably incorrectly called cardiospasm at times, is defined in the *Annals of Surgery* for January, 1925, by Dr. Peter Nicholas Bull of Oslo, Norway, as a diffuse enlargement of the lumen of the esophagus, usually of the lower two-thirds, which exceeds that of any other affection of the

esophagus, and an elongation, presumably congenital, which gives rise to a kinking, the condition never being cured spontaneously or by medical treatment alone.

In a series of 102 cases of megaesophagus reviewed by Dr. Bull, 50 per cent of the cardia were normal, and in the other one-half the lower end of the esophagus showed more or less stenosis and was often hard and contracted.

As to the pathogenesis, much is conjecture. Cardiospasm, with relaxation and dilatation at a higher level, has been primarily stressed. Supporting his theory, for older patients, is the clinical picture of the onset of an attack of obstruction with large, rapid eating, bad mastication, the abuse of alcohol and the ingestion of extremely hot or cold drinks. The mucosal inflammation and ulceration at the lower end of the esophagus, as found in this case, might be considered as the possible origin of reflex spasm, but would more logically appear to be the result of food stagnation that we know was present. Gastric ulcers, gallstones, chronic appendices and gastrocoloptosis have also been observed in conjunction with megaesophagus and suggested as the origin of reflex cardiospasm, but eradication of such conditions where possible, in a number of cases reviewed by Dr. Bull, failed to give benefit, and one gains the impression that they were only coincident.

Furthermore, even in marked stenosis, of corrosive or malignant origin, there never has been reported, according to Bull, such a degree of dilatation. Atony, alone of the muscle, as is rarely seen, with diphtheritic toxemia, should give a thinning of the wall, with dilatation, rather than a thickening of the muscle, as is seen in this condition.

In this case vomiting began at birth. In Dr. Mosher's museum is a typical specimen of megaesophagus occurring in a child that was stillborn. Of 141 cases reviewed by Bull, 82 per cent began to show symptoms between the ages of ten and forty, and the great majority between ten and fifteen.

The most logical explanation of this clinical entity, therefore, appears to be a congenital anomaly. Just as we find congenital dilatations elsewhere; as in megarectum, Hirschsprung's disease, megavesica and hydrocephalus, here also, this organ must have been too long and too wide to accommo-

date itself to the thorax without redundancy and obstructive kinking.

The symptoms and findings are explainable on such a mechanical basis.

It is true there was some evidence of stenosis. But from the time of the first esophagoscopy there were evidences of chronic fibrotic to irritative ulcerative inflammation in the walls of the dilatation. The cardia of this two-year-old child allowed the passage of a No. 16 Tucker bougie on the first attempt, and five weeks later a No. 24, and this moderate amount of stenosis would appear to be no more than would be expected, with the degree of chronic inflammation present.

The treatment, partially successful for these few months, was primarily mechanical and appears to have given the degree of relief that it has because the kink was probably straightened out and the somewhat contracted orifice dilated.

XXXVI.

A CASE OF THROMBOSIS OF THE LATERAL SINUS  
WITH EXTREME RHEUMATOID ARTHRITIS  
(STILL'S DISEASE).\*

BY ARTHUR M. ALDEN, A. M., M. D.,

ST. LOUIS.

R. H., nine years old, admitted to the Children's Hospital February 3, 1925. The parents state that just before Christmas, 1924, the boy had a severe cold, and after a week of coughing, developed what the family physician said was pneumonia. He was in bed two weeks with this. Two days before Christmas he complained of severe earache on the right side, and the day after Christmas the mother noticed that this ear was somewhat swollen and protruded from the head. At this time he had fever, and for four days was in constant pain from this ear. At the end of four days the ear began to discharge and the pain subsided, but the child complained of stiffness in his legs and would not move them without crying out. Throughout January he continued a septic course, with fever from 99 to 104, which alternated with chills and sweats. During this entire time he was in constant pain from the joints of his legs. During the last two weeks of January the fever was much less until two days ago when it came up again to 105. The joint soreness is now much worse, and at the present time also affects the arms.

Examination.—A very much emaciated boy of nine years, acutely ill, lying on his back with limbs drawn up. The child cries out when any of the extremities are moved. The skin is very pale as are also the mucous membranes. The spleen is easily felt and the liver can be palpated four cm. below the costal margin. The musculature of the extremities is extremely wasted. The right arm can be moved slightly with-

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\*From the Department of Otolaryngology, Washington University Medical School, St. Louis, Mo.

out pain, but the child fusses when any of the other extremities are moved. The left ear is normal save for slight retraction and loss of luster of the drum. The right ear canal is filled with thick yellow pus which, when cleaned away, reveals a perforation just behind and above the short process of the malleus. There is a distinct sag of the superior wall of the external auditory canal. The tonsils are small, submerged and definitely infected. The nasopharynx is almost entirely filled with an infected adenoid mass and there is pus on the floor of each side of the nose. The anterior cervical lymph glands on the right side are much swollen and are sensitive to touch, although no fluctuation can be determined. R. B. C. 3,424,000; W. B. C. 9,100. Blood culture "negative." The examination of the eye grounds revealed no abnormality.

Operation.—February 5, 1925. Under gas-ether anesthesia the usual simple mastoid operation was done. The mastoid cells were of the diploetic type. There was some bony destruction around the antrum which was filled with pus and granulations. The bone over the lateral sinus was firm and showed no evidence of necrosis. The lateral sinus was exposed about one cm. from the mastoid tip, and upon the removal of the bone covering the sinus, free pus flowed into the wound. The dura covering the sinus in this region was necrotic and the pus came from the sinus itself. The wound was lightly packed, and after change in toilet the internal vein was exposed in the inferior triangle just external to the sternomastoid muscle. This was done on account of the extreme cervical adenitis in the superior triangle. The vein was picked up and ligated in two places, but not resected, and the incision closed with clips. Returning to the mastoid wound the bone over the lateral sinus for the distance from the mastoid tip to within one cm. of the superior longitudinal sinus was removed. The lateral sinus itself was filled with a partially broken down thrombus and from the region of the jugular bulb free pus was obtained. Free bleeding was not obtained from the posterior end of the sinus, although a probe was passed backward in the sinus as far as the sinus confluens. A gauze drain was placed in the sinus throughout its entire extent and the mastoid wound partly closed with gut and skin clips.



February 6, 1925. The child's general condition is good. There is distinctly less soreness in the joints and the patient appears less toxic. Five hundred c. c. of citrated blood given with very little reaction.

February 7, 1925. Culture from the ear showed staphylococcus aureus. Cultures from mastoid and lateral sinus taken at time of operation showed hemolytic streptococcus. Blood culture negative.

February 9, 1925. The child can now straighten out one leg and the other joints are much less sensitive to motion than before.

February 24, 1925. Child sitting up in bed and can move all joints without pain.

February 26, 1925. Tonsillectomy and adenoidectomy under gas, uneventful.

March 3, 1925. Child discharged from hospital in good condition.

FRISCO BUILDING.

XXXVII.

SOME OF THE PROBLEMS OF THE HARD OF HEARING AND THEIR SOLUTION.\*

BY HAROLD HAYS, M. D.,

NEW YORK CITY.

A subject of this kind is so large that it would be impossible to enter into a general discussion at this time. Within the past two decades, great strides have been made in the management of the deaf, but I regret to say that we are far from that millennium where we can promise that we can cure deafness. This is not due entirely to a lack of desire on the part of medical men, but is due, to a great extent, to two factors—first, the negligence of the patient and, second, the impossibility of definitely interpreting the pathologic condition behind the ear defect. For example, a patient presents himself with a hearing defect which shows plainly that the internal ear is involved. The question arises, is there not some middle ear condition which is aggravating the internal ear trouble? Is it not possible that the middle ear condition can be improved and even arrested? And is it not utterly wrong to state to such a patient that they cannot be improved because an internal ear pathology has been found?

The greatest encouragement has been found in the interpretation of the causes of deafness and in the management of the average case which is amenable to treatment. The time worn use of the inflation method by the catheter or by Politzerization has practically been discarded and the intelligent otologist of today will not be satisfied until he has made a satisfactory hearing test, has examined into the causes of the ear condition and has treated the pathology as he has found it. He has been greatly aided by having at his disposal various electrical devices, such as the nasopharyngoscope and the auro-

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\*Read before the American Academy of Ophthalmology and Otolaryngology, annual meeting, Chicago, October 20, 1925.

scope. He is now able to examine the pathology of the nasopharynx with as much exactitude as he is able to examine the oral cavity, and he is also able to interpret the conditions of the drum membrane so that he is not content with a hasty examination and the assertion that the drum is retracted. Even in cases in which there seems little doubt that there is a nerve lesion, I have seen the hearing improve fifty per cent by proper treatment of the tubal mucosa and by establishing the patency of the eustachian tube. Another factor of great importance which was too long unrecognized was the effect of various general abnormalities on the hearing mechanism. The otologist of today realizes the importance of proper inquiry into various ailments which are far removed from the ears and, in the majority of instances, he will refuse local treatment unless the patient receives the proper care of his other infirmities. I have seen many a case of deafness improve after a gastrointestinal condition was cleared up and I have known of numerous instances where the patient's hearing defect improved considerably after the general resistance was brought up to par.

But, to my mind, the one factor which will count for more than anything else in the advancement of our knowledge of this subject is the awakening of interest in it on the part of the otologists. I can well recall the hopeless position that most of us were in until recent years. The deaf patient was considered more or less of a nuisance—at least, one felt that very little could be done for him if he did not improve after one or two inflations with the catheter. It was seldom that the otologist took the time and the pains to explain the condition to the patient and to put him in the way of making his life more useful and happy. One often heard the bromidic remark, "I fear your condition is progressive and little can be done for you." This often meant mental suicide to the patient. Or again, "You have otosclerosis." Never was a term more loosely used. I believe that Dr. Pierce once made the remark that we have yet to see the first case of otosclerosis in this country which was proven to be such on autopsy. Sadder than this is the fact that many a case which was diagnosed as otosclerosis on hasty examination responded to treatment at the hands of another otologist. I have seen many a case

of relaxed ear drums or pocket handkerchief deafness which was wrongly diagnosed as otosclerosis.

Conjecture as we may, there is no doubt that the psychologic state of the patient, at any given time, has a great deal to do with the ability to hear. Given an individual patient, he will invariably hear better when he is in an exalted state of happiness than he will when he is in a state of depression. I have in mind the case of a young woman who has been a patient of mine for many years. There is no doubt that she has a nerve deafness which is aggravated by changes which take place in her middle ear. When Miss A. first came to me, it was impossible to obtain any improvement in her hearing. She was studying hard to become a teacher, desired to mask her hearing defect and was in a continuous state of mental and physical depression. Her eustachian tubes were well open and no definite pathology could be found in her nose or throat. After a number of years, she succeeded in getting herself placed as a teacher, her home surroundings improved, she became more mentally alert, she adopted a different attitude toward her hearing defect, with the result that her deafness remained stationary. Last spring her hearing was tested on the audiometer and disclosed a defect of fifty per cent in her right ear and over forty per cent in her left ear. She left town for the summer. A test of her hearing was made at the offices of the Western Electric Company and in our office on one of their audiometers, and an improvement of over ten per cent was found in both ears. No treatment had been given her in the interim but her general health had improved. This is only one of many instances where the proper handling of the patient, from the psychologic point of view, has meant an improvement in hearing.

Unfortunately, as our knowledge of the pathology of deafness increases and our ability to help the deaf in various ways becomes more evident, we find that the general deaf public become more gullible. The main reason for this is that the treatment of the deaf has become more popular and the charlatan has become more intelligent in his means of approaching the deaf. It is seldom that he attempts to use time worn instruments which will be a cure all. Today he adopts the so-called scientific method and poses as a scientist. He may be

found among the ranks of the "outsiders" among those who suddenly conceive a form of treatment which would be farcical if it were not that so many hopelessly deaf people are fooled. He may be found in the ranks of the pseudoscientists, chiropractors, naturopaths, osteopaths, Christian Scientists, etc.: and more unfortunate still, he may be found in the ranks of the ethical medical profession. It is not my purpose to dwell upon any of these especial methods of treatment nor to attempt to expose any of them. But our more definite interpretation of hearing defects, as was previously pointed out, has shown us that this infirmity is caused or at least the infirmity increased by a multiplicity of conditions, many of which are local and many of which are general.

One may consider that the charlatan is the man who refuses to allow his type of treatment to be tested out by other scientific men so that it may have the stamp of their approval.

The X-ray treatment of deafness has been given a fair trial and, I do not hesitate to say, has not met with any marked success. Finger manipulation may have its place in the osteopathic armamentarium, but I again do not hesitate to say that it has not met with great success except in the hands of those who are born with special fingers and the mind to guide the finger into the right places. Nasal operations, of one kind or another, have been advocated as a means of improving the hearing ever since the otologist has been able to wield a knife within the nose. Evidence points to the fact that any such improvement is transitory, seldom permanent. Theorize as we may, we are not able to overcome definite connective tissue changes by operating upon an extraneous part. The massaging of a spine may stimulate the nerve endings, but it is more likely to suggestively stimulate the digestion and the cerebral cortex so that a person changes his thinking mechanism. The same may be said of psychoanalysis and Christian Science. Fortunately the advocacy of wearing artificial ear drums for every form of deafness is going out of vogue.

To this list of special types of treatment which have been much heralded may be added the claims of those who advocate the use of certain apparatus, such as the numerous types of vibrators, the concussors, various forms of electric treatment,

such as ultraviolet rays and diathermia. I do not by any means wish to intimate that these types of treatment (including those which are most universally condemned) have no special uses, and I agree that, in certain instances, marked improvement may be obtained, but one must insist that that one type of treatment must not be the only one used. I am a firm believer in the various forms of electro- and mechanotherapy, but I have yet to see one single case in which improvement took place by the application of any special method of treatment.

The reason for the charlatan's success is almost self evident; the removal of the evil is not so simple. His success depends first on the gullibility of the human animal, and secondly on the intense desire of the deaf individual to rid himself of an infirmity which he at all times wishes to hide. He always feels that there is someone in this world who ought to be able to help him, and having met with discouragement at the hands of the scientific otologists, he feels that perhaps some new man who promises much will be able to do more than the man who has spent all his life in trying to solve this problem. No more illuminating evidence on this subject could be presented than that given by Dr. Harold Rypins in his classic paper before the American Federation of Organizations for the Hard of Hearing in Minneapolis last June.

Dr. Rypins' suggestion for a cure is education of the deaf people themselves. He wishes to have anyone who claims a cure for deafness to be brought before a tribunal—that is, before the various bodies of the deaf who are working for the deaf. He wishes an index kept on each and every form of treatment and confessions of deaf people so treated kept on record. If there is anything in any form of treatment, no matter by whom advocated, we want to know it. If there is nothing in their forms of treatment, we want the deaf to know it. The medical men can rail against these pseudo-scientists forever and anon; it will do little good. But once the deaf themselves refuse to go to these individuals, once they spread the nakedness of the truth about, then only will these individuals die of starvation and try their misguided intellects in other fields.

In the not far distant past, the handling of the deaf patient was left entirely in the hands of the physician. The awakening began with the formation of various organizations of the deaf. This has meant far more than we can imagine at the present day. I am happy in the feeling that I organized the first League for the Hard of Hearing in this country; but if it had not been for the devoted interest of the late Edward Bartlett Nitchie and the Alumni Association of the New York School for Lip Reading, it would have been impossible to have obtained the wholehearted cooperation so necessary in a movement of this kind. The prime object of the New York League was social service among the deafened. No sooner had we begun this work than we found that this was but a small branch of it. Numerous people needed instruction in lip reading; many of them had to be mentally rehabilitated; some of them needed financial aid. As soon as we began to work on these problems, we found that we needed a consulting board of otologists who would be able to separate the deaf people who could be helped by medical means from those who could not. And finally, within recent years, we have launched into the field of prevention and have begun to work on the children at an age when they can be improved and sometimes cured. Our figures, after working on hundreds of school children during the past few years, agree with those of others and, briefly, we have come to the following conclusions: (1) That about three per cent of school children are suffering from ear conditions which interfere with their school work; (2) that the cases equally divide themselves between those who are suffering from suppurative conditions and those who are suffering from so-called catarrhal otitis; (3) that many of these children have not had their tonsils and adenoids removed or inflammatory or suppurative conditions of the nose attended to; (4) that many of these children have a tendency toward a relaxed ear drum because they do not blow the nose properly; (5) that a majority of the suppurations can be arrested by proper treatment and without performing a radical mastoid operation; (6) that 80% of the children who are suffering from catarrhal conditions can be improved or cured by simple Politzerization. These figures are encouraging and demonstrate that it is most necessary to resort to a campaign

of education until it becomes the rule for every school child to have his hearing tested at regular intervals.

About five years ago, the American Federation of Organizations for the Hard of Hearing was formed with Dr. Wendell C. Phillips, president-elect of the American Medical Association, as the first president. Mainly through his efforts and the efforts of the loyal members of various local organizations, we were launched into almost immediate success. I had the pleasure of serving as the second president to be succeeded by that most conscientious otologist, Dr. Gordon Berry, who has placed the organization on a true scientific basis. We are fortunate in having as our president-elect the president of our Academy, Dr. Horace Newhart, who is sure to carry the work on toward its ultimate success. Both Dr. Phillips and I are proud of the fact that we have been succeeded by men who have the ambition, the energy and the enthusiasm to put over any project in which they are interested.

Why should the Federation receive the support of the medical profession? Why should every otologist know of its work? In what way has the Federation been of help to the otologist? The reason why we, as medical men, should support it is because it is doing its utmost to advance the welfare of the deaf, medically, mentally, economically, and sociologically. Every otologist should know of its work because the Association is trying in every way to simplify the mental and physical condition of the deaf, and thus make the work of the otologist much easier. In many other ways has the Federation been of help to the otologist. First, it has attempted to steer the deaf away from the charlatans. Second, it is collecting statistics to show that deafness is preventable so that the deaf child will be brought to the specialist at an earlier age. Third, it has compiled for you the names and addresses of the lip reading teachers throughout the country so that you will know to whom to refer your patients. Fourth, it is now attempting to inform you as to the worth of the various hearing devices being advertised. Fifth, it is attempting to standardize hearing tests so that all of us will make tests in the same way, and last, it has formed a general scientific committee composed of physicians, physicists, educators, etc., who, no doubt,



will present some amazing data to you in the near future. Moreover, the Federation has been instrumental in the formation of committees dealing with the welfare of the deaf in our various national societies, with the result that the work of the Federation has been indorsed by the House of Delegates of the American Medical Association. Surely such work should meet with your approval and indorsement and it should be the ambition of every otologist present to associate himself with one of the local branches of the Federation.

Summing up the advances that have been made in the treatment of the deaf, we may conclude as follows:

1. The attitude of the otologist toward his deaf patient has changed.
2. More interest is taken in the prevention of deafness.
3. Psychologic treatment is of the utmost importance.
4. The elimination of the charlatan will depend on the success of a campaign of education against him.
5. The lay organizations for the deaf, such as the American Federation of Organizations for the Hard of Hearing, are helping a great deal in solving the problem of the deaf.

22 WEST 74TH STREET.

XXXVIII.

SYPHILIS WITH SPECIAL REFERENCE TO ITS  
ORAL MANIFESTATIONS.\*

BY WILLIAM B. TATUM, M. D.,  
BROOKLYN, N. Y.

Syphilis is a disease produced by the spirocheta pallida and manifests itself in such a varied manner that it would be futile for me to attempt to discuss this condition, other than the lesions which are of particular interest to the oral surgeon. During the past six years I have had the opportunity to observe and treat conditions produced by the spirocheta, both in this country and in Europe. Syphilis of the oral cavity manifests itself both after congenital and acquired infection. The congenital type is what the word implies, that is, infection as transmitted to the child in utero. The first manifestations are usually observed between the fourth and eighth week with nasal catarrh, discharge from the nose, which is often purulent or bloody, oftentimes there are present fissures in the angles of the mouth, known as rhagades, and the mouth presents a puckered appearance. Frequently the condition progresses until there are ulcerations of the palate with necrosis of bone leading to the depression at the root of the nose commonly spoken of as saddle nose, which, when seen, should never be mistaken for any lesion other than syphilis, except depressions due to injury, poor surgery or rare infections of nasal bone. The second type of manifestation is bleeding from the mucous membrane of the oral cavity. This is not a frequent finding, and therefore should not be considered syphilitic in origin unless there are other manifestations to warrant such a diagnosis, as cutaneous rash, or history of parental infection. Many children suffering from syphilis of this type die in infancy. The second most frequent period begins about the age of permanent dentition, at which time there may be husky voice, and malformations of the teeth, such as irreg-

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\*Read before All-Surgery Section, Second District Dental Society meeting at Brooklyn, N. Y., February 26, 1925.

ular, peg shape, poorly enameled, widely separated teeth. These need no further discussion as you are all familiar with such pictures. At about this age and up to puberty there is also commonly noted Hutchinson's triad, that is, notched teeth, interstitial keratitis and deafness. These are a few of the common lesions observed, but no doubt are infrequently seen by the oral surgeon. The stigmata which you should be on the lookout for are the scars of a once active lesion. After the age of puberty is the time that the oral surgeon comes in contact with the individual. He should always be on the lookout for the following:

Radial white lines about the month, which are the results of rhagades.

Both peg and Hutchinson type teeth.

Necrosis of both hard and soft palate.

Perforation of the nasal septum and saddle nose.

There may be also gummatous formation of any part of the oral cavity. This condition is rare and I will not dwell upon it. The acquired infection is arbitrarily divided into three stages: primary, secondary and tertiary. However, one must not carry the idea that this classification is clear cut. One commonly observes the primary and secondary lesions at the same time. As a rule, however, the secondary lesions appear about the time the primary sore has healed. Then, too, in some instances the secondary lesions are associated with manifestations which are interpreted as tertiary lesions.

The primary sore, known as hard chancre, has an incubation period of about twenty-one days, but may be as long as four weeks. The situation of the primary sore is variable. About 90% occur on the genitals, the other 10% occurring on fingers, toes, breasts, eyelids and nose, but particularly the lips, tongue and tonsils. Therefore, I think it is safe to say that from 8 to 9% of all syphilitic lesions have the primary sore in or about the oral cavity. Based on my personal experience, it would seem that the extragenital chancre is increasing in frequency.

Mode of infection: The entrance of the spirochete is through an abrasion of the skin or mucous membrane. The sore begins as a small granuloma, which later becomes an erosion, due to the pressure; necrosis with a copper red, boiled ham base; the

edges are firm and well defined, and in many instances the sore is cartilaginous in consistency, hence the term hard chancre.

Spirochetes may be demonstrated by the dark field method, from fresh irritation serum obtained from the floor and edges of the ulcer. By the dark field we mean an instrument which has a completely dark center with the reflected light coming from the periphery. It is likened to a particle of dust shown in a ray of light in a dark room.

The most common sites of the mouth lesion, in order of frequency, are the lips, tongue and tonsils; however, the sore may be on any part of the mucous membrane. There is also a regional glandular involvement so that if one palpates in the region of the angle of the mandible there will be felt definite small, fairly firm, freely movable, painless, enlarged lymph nodes. The absence of tenderness, both in the primary lesion and in the enlarged lymph node, is quite characteristic of syphilis. There is no inflammatory redness over these glands. The disease at first is a local condition but does not remain so for a very long period of time, probably becoming systemic very soon after the appearance of the chancre. As before mentioned the first evidence of this is the regional glandular enlargement, later the general glandular enlargement and still later the cutaneous rash which occurs between the eighth and ninth week and is often associated with fever, afternoon headaches, general malaise and loss of appetite. The mode of entrance of the spirochete into the blood stream is as follows:

They penetrate from the tissue of the primary sore to the lumen of the gland and through the lymphatic stream to the blood of the vena cava.

A few spirochetes may also enter through the blood vessels to the blood stream, but not as many enter this way as by the lymphatic system.

Coincident with the cutaneous rash there appears a very important manifestation, the markedly injected pharynx, followed by mucous patches of the mouth and throat. This involvement of mouth, pharynx and larynx necessarily involves the vocal cords, producing typical sore throat and husky voice. During the second stage of the disease the most significant

lesion is the so-called mucous patch. This lesion presents a variable picture, having as a constant factor hypertrophy of the mucous membrane, that is, the lesion in the early stage is always raised above the surrounding mucosa and looks as though silver nitrate had been brushed across the surface. At a later stage of their development they show a superficial necrosis appearing grayish white. There is always present a zone of hyperemia at the edge of the necrotic area, presenting a shallow punched out ulcer. These mucous patches are always associated with the cutaneous rash, in fact, they are nothing but a rash of the mucous membrane. The mucous patch is very, very infectious and should be handled with great care. When healing takes place there is seldom a trace of the destructive process, the mucous membrane resuming its normal appearance. A little later we see the dry moth-eaten hair, and patches of baldness over the head, commonly known as alopecia. We also see the pigmentation of the skin of the neck, particularly in the female, due to lack of growth of hair. The tertiary lesions usually appear after the first year of the disease; however, they may appear as early as six to eight months after infection. They are the least infectious, the most varied and are the type of lesions that Sir William Osler had reference to when he said: "Know syphilis and the rest of medicine will be added unto you." The common manifestation of tertiary syphilis is the gummatous formation. By that we mean the circumscribed mass of new tissue composed of many small round cells and an occasional giant cell. As the mass continues to grow, the center becomes necrotic because of the accompanying endarteritis and pressure on the blood vessels by the hyperplastic tissue. At a later stage the gumma is surrounded by fibroplastic tissue, which is nothing but Nature's method of limiting its growth. During the early stage of gummatous formation the spirochetæ exist in the walls of the blood vessels and in the surrounding cellular element. Later when degeneration occurs the spirochetes disappear; therefore, when one observes the tertiary lesion of the mouth, which has become necrotic and ulcerated, the spirochete can no longer be found, thus accounting for the noninfectious nature of the lesion. They may appear in any tissue of the body, the mouth being no excep-

tion to the rule. In and about the oral cavity we have mucous membrane, muscle cartilage and bone as the important structures. The gummata of bone involves the maxilla much more frequently than the mandible. The process here is no different than that previously described, that is, necrosis takes place and extends through the structures covering the bone discharging the necrotic center into the oral cavity. Subsequently the mouth organisms invade the cavity of the gummata, thus adding insult to injury. From this secondary infection there is death of bone. With sequestration this process may be limited or in some cases fairly extensive, involving the entire palate. If the process be extensive enough teeth will come away with sequestered bone. After Nature has limited the destructive process, repair begins. This repair never fills in the defect produced by the destructive lesion, but the remaining structures are covered by fibrous tissue and ingrowing mucous membrane; therefore, one may see a varied type of destructive defect of the oral cavity. According to the stage of the lesion one sees exposed bone change, sloughing cavities, healing processes or the entire healed area with the accompanying defect. The tongue, which is frequently affected, produces the same type of lesion, that is, gummatous formation or ulceration. In the tongue we often see fissures which represent the healed process of small localized gummata. There are many other rare tertiary lesions of the oral cavity which I will not discuss. It is not difficult for one to observe an abnormality of the oral cavity, due to disease, but in many instances it is extremely difficult to determine just the exact nature of the process.

The differential diagnosis of syphilis and "syphilislike" conditions of the mouth often entails many highly specialized examinations together with much laboratory data. I will attempt to point out the important differential points of diagnosis. Of the congenital lesion of the oral cavity, the most common ones to differentiate are the results of some previous operative procedure, noma (butterfly shaped necrosis about the mouth and face following exanthemata, scarlet fever in particular), and leprosy with nasal involvement. The postoperative defects can be determined from the history. The destructive processes following cancrum orum (noma) may represent many difficulties.

It is usually differentiated by very careful history and Wassermann reaction. Leprosy is so rare in this country that it need not be considered. Of the primary lesion one must consider carcinoma, tuberculosis, Vincent's angina, canker sores, herpes labialis and trauma. In carcinomatous lesions the individual is usually past middle age, frequently there is glandular involvement, the glands being stony hard in consistency. In the absence of positive Wassermann reaction, history of exposure, negative dark field examination, etc., one should always take a small section for pathologic examination. What applies to carcinoma also holds true of tuberculosis, with the exception that in most cases there is present an active tuberculous lesion elsewhere in the body, particularly in the lungs. In carcinoma and tuberculosis, the ulcers are commonly of long standing. Vincent's angina may represent both primary and secondary lues. The lesion that simulates a chancre usually appears in the tonsils. The ulcers of Vincent's are painful, punched out and more destructive than the luetic ulcers. The regional glands are also enlarged and tender, while in syphilis they are painless. The dark field examination and stained slide in Vincent's reveals the long typical spirochete which has not the rotary movement, is large and its coils less sharp. The spirochete of Vincent's is usually accompanied by the fusiform bacillus. The spirocheta pallida is usually six to fourteen microns in length and one-quarter micron or less in breadth and have six to twelve sharp spiral coils resembling a corkscrew, and its movement is rotary upon its long axis.

Canker sores are small, flat, trough shape ulcers, with a yellowish white fibrinous coating and an intensely red border of inflammation. The onset is sudden, the ulcer is very painful and we do not have glandular involvement.

Herpes labialis usually occurs as the result of high temperature. It appears as small vesicles, the contents of which are first clear but soon coagulate. The surrounding skin is slightly reddened. One should not mistake this, but when a single herpetic lesion is seen, when the roof, the vesicle and the coagulated contents are cast off, it may resemble the primary lesion of syphilis.

Trauma with secondary infection may resemble primary sore, but the history usually rules out syphilis. All of the above

mentioned conditions may also occur in patients who are, or have been, infected with syphilis. The Wassermann reaction is not of much value in the primary syphilis, as at this time the disease remains more or less a local condition. However, the dark field, pathologic sections and stained slides will make the diagnosis comparatively easy. Of the secondary lesions of the oral cavity one must think of carcinoma, tuberculosis, Vincent's angina, canker sores and trauma, which have already been described, the only difference from that resembling the primary infection being that they are of longer standing, and, as more ulceration has taken place, they look more like deep mucous patches. The most common ones to be differentiated are those produced by large doses of mercury. In mercury stomatitis, the mucous membrane of the mouth is reddened and swollen. The edge of the gums has a bluish tinge, is slightly necrotic with a purulent coating, and, on pressure, pus comes from around the teeth. This is a mild form. If severe enough, ulcers of the tongue are produced, corresponding to the adjacent teeth. These ulcers are due to pressure of the swollen mucous membrane against the teeth. The breath is very foul and there is marked salivation. We may also have a mercurial angina which looks like diphtheria, but this can be ruled out by cultural methods. The oral manifestation following mercurial medication is very apt to be confused with syphilitic mucous patches, and this stomatitis most commonly occurs in patients who are receiving treatment for syphilis.

Antipyrin rash of the tongue is sometimes seen. This comes on in a few hours after taking the drug. It produces a sharp, burning sensation, with reddening and swelling of the mucous membrane. The tongue is swollen and rough, vesicles form, there is excoriation of the tongue, and it is deeply furred and may have the appearance of secondary or even a tertiary syphilitic mouth lesion.

Of the other, less common conditions resembling secondary mouth syphilis are lichen ruber, fungous angina, geographical tongue, leukoplakia of the tongue and exudative erythema. These are very rare and need not be described. Acute follicular tonsillitis may sometimes be confused, but you are all familiar with such an affection. In tuberculous ulcers of the hard and soft palate and pharynx we see an intensely red, rough milary



surface with steep edge ulcers. Many tuberculous nodules may be seen studded about the area, and tubercle bacilli can be recovered from them. These ulcers may progress until they are very deep and become very painful.

In any of the above mentioned conditions, by a careful history and physical examination of the part, pathologic examination, cultures, stained slides, dark field examination and Wassermann reaction, the diagnosis can be established. Of the tertiary lesions we must think principally of carcinoma, tuberculosis, sarcoma, actinomycosis, echinococcus cyst and fibroma of the tongue. These can hardly be differentiated from gumma by their appearance except by pathologic sections and repeated absence of positive serum reaction.

I think the best way to leave a lasting impression of these conditions is by showing you a few photographic plates taken from the patients and at the same time discuss them informally.

2 SCHERMERHORN STREET.

## SOCIETY PROCEEDINGS.

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### THE CHICAGO LARYNGOLOGICAL AND OTOL- GICAL SOCIETY.

*Meeting of Monday Evening, December 7, 1925.*

THE PRESIDENT, DR. HARRY L. POLLOCK, IN THE CHAIR.

#### CLINICAL MEETING.

#### **Osteomyelitis of the Superior Maxillary Bone. Orbital Cellulitis and Abscess.**

DR. C. F. YERGER presented a patient, 17 days old, who was admitted to the Cook County Hospital, Department of Ophthalmology, on Dr. George Suker's service, with the diagnosis of a retrobulbar tumor of congenital origin.

The mother stated that the baby was born of normal delivery and had been in good health until four days before bringing it to the hospital, when she noted that the child's right eye was swollen and pushed forward. This became worse in the course of a few hours, and by the following day the proptosis had become still more marked, and there was associated a discharge of pus from the right nostril.

When the baby was 14 days old the mother noted that it had a canker sore in the mouth. A history of trauma, of diseased or dirty nipples, or of the presence of a breast abscess could be excluded, although the mother developed an abscess of the breast, subsequent to the time it was discovered that the baby had a discharge of pus from the mouth, when the baby was 18 days old. The mother's breast was infected by the baby and not vice versa.

Examination showed a very marked proptosis of the right eye with conjunctival injection but no discharge. Transillumination of the eye was normal. There was a profuse discharge of pus from the right nostril and mouth. There were two fistulae along the right alveolar ridge at the level of the first and second molars, which exuded pus. Pressure over the

right cheek increased the drainage from these openings and from the right nostril, and also exaggerated the proptosis, showing that a communication existed between these regions. The right side of the hard palate was swollen and flush with the alveolar ridge, obliterating the normal concavity of the palatal arch as far as the median line.

Dr. Suker diagnosed the case as one of suppurative nasal sinus disease. Numerous attempts at roentgenography failed, due to the baby moving.

The fistulae along the alveolar ridge were probed but did not seem to connect with the orbit. After the extraction of one of the unerupted teeth, which presented at the orifice, a probe was readily passed from the alveolar orifice to the orbit. The temperature was septic during the baby's first two weeks in the hospital, the purulent discharge from the nose and mouth continued and the proptosis was unchanged. The treatment consisted of frequent cleansing of the discharge from the nose and mouth, and the baby was taken off the breast and the bottle substituted.

In the beginning of the third week, hoping to increase drainage from the orbit, the fistulae in the alveolar process were curetted and extended upward toward the floor of the orbit. In doing this several tooth germs were removed. The proptosis gradually became less marked, but was still somewhat in evidence after a month's residence in the hospital.

During the height of the compression upon the eyeball by the orbital cellulitis and the presence of a most marked proptosis, a choked disc resulted, which later was followed by atrophy of the optic nerve. (Suker.)

At the time of presentation, three months after the baby entered the hospital and two months after she was discharged, there was present a slight amount of proptosis and a purulent nasal discharge occasionally. Another attempt was made to obtain suitable roentgenograms, but Dr. Matthews of the Roentgenologic Department was unable to state definitely whether or not an osteomyelitis of the superior maxillary bone was present, but did exclude the nasal sinuses as a cause of the disease.

**Progressive Osteomyelitis of the Frontal Bone.**

DR. YERGER'S second case was a male, age 51, who entered the Otolaryngologic Department of Cook County Hospital on September 18, 1924.

Six months previously he developed an acute swelling over both maxillary and frontal regions, which was followed by a fistula on each side of the bridge of the nose. One month later an abscess appeared over the left supraorbital region, followed after one week by an abscess over the right supraorbital region, both of which opened spontaneously and discharged pus. He had a positive Wassermann reaction and had received fourteen injections of arsphenamin and a course of mercurial inunctions. He had had a saddle nose deformity for 24 years, following a nasal operation.

Examination showed hyperemia and induration over both maxillary and frontal regions with discharging fistulae, one on each side of the bridge of the nose and in each supraorbital region. The discharge was a fetid, creamy pus. The right eyelids were swollen and the eye closed. The blood Wassermann reaction was negative.

A diagnosis of chronic osteomyelitis of the frontal bone, due to frontal empyema or syphilis, or both, was made.

Operation: A bilateral suprarobital incision was made, connecting over the bridge of the nose and extending laterally upward toward the vertex. Necrotic portions of the frontal nasal and vomer bones were removed, and a modified bilateral Killian frontal sinus operation was performed with drainage of extradural abscess. A pachymeningitis externa was present, and the dura was covered with a thick layer of edematous granulations, which were removed.

Ten days later a fluctuant swelling presented itself over the upper part of the forehead, the sinus over the left midfrontal region opened and one ounce of thick, creamy pus was evacuated. Twenty days after operation a sequestrectomy was done because of positive roentgenographic evidence of bone sequestra. The old incisions were reopened and extended upward toward the vertex and several sequestra and granulations were removed.

Bacteriologic examination showed that the pus obtained at operation contained Gram positive diplococci in chains.

Pathologic examination: "The two pieces of frontal bone removed at operation were 7 by 5 and 4 by 5 cm., the outer surface was spongy, worm eaten and covered by greenish yellow purulent material. The bone substance is dense and not at all spongy, as suggested by the outer surface, and cannot be broken. Pathologic diagnosis: Chronic osteomyelitis with caries and sclerosis of the frontal bone."

Surgical diagnosis: Progressive osteomyelitis of the frontal bone with extensive syphilitic necrosis of the frontal, nasal and vomer bones and the cartilage of the nasal septum; extradural abscess and pachymeningitis externa.

Following the second sequestrectomy the patient made an uneventful recovery and had remained well for 15 months.

#### **Progressive Osteomyelitis of the Frontal Bone.**

DR. YERGER'S third case was a boy, aged 12, who entered the Otolaryngologic Department of Cook County Hospital on February 12, 1924.

Five days previously he had a right external frontal sinus operation at another hospital. For four days following the operation he improved, but on the fifth day became delirious.

Examination showed a poorly nourished boy in a delirious and stuporous condition, from which he could be aroused into consciousness for a few minutes and would then relapse into his former state. There was present a soft edema involving the scalp over the frontal and parietal areas, extending down to the zygoma and involving both eyelids. There was proptosis of both eyes and bilateral chemosis. The white blood count was 27,000. Spinal fluid examination showed 52 cells, mostly lymphocytes.

Diagnosis: Acute frontal sinus empyema, orbital cellulitis, cellulitis of the scalp and recent operation upon the right frontal sinus. A diagnosis of acute toxic meningoencephalitis was made upon the spinal fluid findings and his stuporous condition, together with the absence of positive findings in favor of meningitis or brain abscess.

Operation: Incision along both eyebrows and across the glabella. The anterior plate of the right frontal sinus had been removed and the sinus was found full of thick, creamy pus. The anterior plate of the left frontal sinus was removed and

it likewise was found full of pus. Both nasofrontal ducts were enlarged by breaking down the anterior ethmoids, and rubber drainage tubes were inserted. The wound was left widely open with gauze pack in the wound. Cultures of the pus showed staphylococcus aureus.

The following day he was wildly delirious and had an axillary temperature of 101 degrees. Three days later his sensorium became clear and his cerebration good. Nine days after operation he developed an abscess in the right pectoral region, which was opened and drained. He continued to have a temperature of 1, 2 or 3 degrees for a period of twenty days following operation. After a residence of two months in the hospital he was discharged and treated in the outpatient department, but was readmitted two months later on account of continued suppuration and fistulæ formation due to the presence of sequestra.

A sequestrectomy was performed and the old incision was reopened, the skin flap being carefully dissected from the dura. A small cavity containing pus was found on the right side, external to the dura. The two fistulæ in the right and left supraorbital regions were curetted free from granulations, two large bone sequestra were removed and the wound packed with gauze.

The discharge of pus continued from the edge of the wound in the midline over the glabella and through a fistulous opening in the midline within the hairy scalp. Pressure made on the superior swelling caused pus to flow from the inferior fistula. A roentgenograph showed the presence of sequestra, necessitating a second sequestrectomy four months after the first.

At this operation two thin bony sequestra, 1 by 3 cm., were removed. The superior fistula communicated with a circumscribed osteomyelitic area 2 by 2 cm., which was filled with granulations. The carious bone was thoroughly removed. Normal bone separated the interval between the area of bone necrosis and the previously operated area of osteomyelitis to the extent of 4 cm., proving that the process, in this area at least, was due to a localized hematogenous infection and not to involvement by contiguity, as had occurred in the area previously operated on. The operative technic differed from the two previous ones in that a horseshoe flap, with the base down and its

convexity situated well up within the hair line of the forehead, was made in order to be able to follow the osteomyelitic process into the vertex region. The inner surface of the flap and the two fistulous openings contained much edematous granulation, which was removed. The pericranium was sutured with catgut and the skin closed with silkworm gut. Gauze drainage was provided for through the two fistulous openings, which were enlarged.

A roentgenograph, taken one month later, showed shadows suggestive of sequestra in the frontal region at the hair border and above the root of the nose. Six weeks later, two sequestra the size of the little finger were removed through the superior fistula, from which pus had been discharging. Subsequently there had been no recurrence of sequestration and the patient had remained well for twenty-two months.

#### DISCUSSION.

DR. OTTO STEIN, discussing the first case, said that he saw an almost identical one in a child two weeks old. Following a circumcision by a general surgeon, the child developed a generalized infection, with a discharge of pus from the right nostril and numerous fistulae in the superior maxilla on the right side. They finally succeeded in clearing up the infection.

DR. GEORGE W. BOOT had seen several cases similar to the first, but most of them had followed infection from the ethmoid instead of the maxillary sinus. One patient was a young man of about 20, who had a sore throat and some rash. Later he developed a great deal of swelling of the alveolar process and discharge from the right maxillary sinus. He was sent to the hospital and the sinus was opened. One tooth was very loose and was removed and the infection cleared up.

He thought the two last cases were entirely different in type and that Dr. Yerger should be congratulated on the recovery, particularly of the boy. The case of the man was not so serious and the outlook was very much better.

DR. JOSEPH C. BECK thought the important thing in the first case was the future development, because there would be a hemifacial atrophy, stopping development. The dental process would have to be carefully considered, as the teeth had been

lost through the operation. In a similar case under his care the patient was two months old, and as an end result retraction of the eye occurred and quite a bad deformity resulted. He believed most of the cases were embolic, but some claimed they were due to forceps delivery.

DR. HARRY L. POLLOCK, discussing the first case, called attention to the controversy which had recently appeared in the *Zeitschrift für Ohrenheilkunde*. There had been much discussion between Paunz of Budapest and Körner of Rostock as to the etiology in these cases. Paunz claimed they were all due directly to antrum infection, while Körner claimed they were primarily due to infection of unerupted teeth and cited several cases in which the antrum was perfectly normal. From their discussion one must draw their own conclusions.

Discussing the last two cases he mentioned the case of a woman who was admitted to the North Chicago Hospital the day before, with a history of pain over the right eye, of six months' duration. She had received local treatment from her home physician until about one month ago, at which time the eye began to swell and great quantities of pus came from the nose. She was treated by suction and warm applications until the swelling of the eyelid became worse. Upon examination they found the eyelid greatly swollen and edematous. The patient was presbyopic, but vision was fairly good. They aspirated the pus from the nose for culture. The roentgen examination showed osteomyelitis of the entire frontal bone on both sides, and the frontal sinuses were practically obliterated. They had operated that morning. There was an area the size of half a dollar in which the dura was exposed, filled with granulation tissue, and above this area a sequestrum which was freely movable. There was no intracranial involvement at the time of operation, but with the large dural exposure they had great fear of mortality because the disease was progressing. They had put in drains and left the wound open. The infection could have occurred from the frontal sinus, but they believed it was on a syphilitic basis, although they had not yet received the report on the Wassermann test. They thought the infection had probably originated in the ethmoid and extended upward. The acute infection was of four weeks' duration.



Later report from microscopic specimen proved it to be a squamous cell carcinoma. The Wassermann was negative.

DR. ROBERT H. GOOD related a case of necrosis of the frontal bone, with a large swollen mass in the center of the forehead which had been present for six months. It changed in size occasionally and pus discharged from the nose. He did an intranasal frontal sinus operation and then could squeeze the mass, and pus would come down into the sinus and then into the nose. He instructed the patient to exert pressure on the mass by pressing the forehead to evacuate the pus, which he did for several months, but this did no good, so they decided to operate more radically. At operation they found a conical shaped necrosed area of perfectly green color in the center of the forehead. This entire area was removed to the dura, but the patient afterward developed osteomyelitis and died. He thought they should have drained the necrosed area instead of removing the bone.

DR. CHARLES H. LONG congratulated Dr. Yerger and complimented him on his courage in following up the cases. In London, several years ago, Dr. Long saw several cases of osteomyelitis and their treatment was so unsuccessful that he made no attempt to follow them up. Their mortality was almost 100 per cent, no matter what the cause.

DR. C. F. YERGER (in closing) said he was aided in his diagnosis in the first case by an article which Dr. Posey of Philadelphia published in the Journal of the American Medical Association in 1912. In that article he stated that in infants under two years of age osteomyelitis of the superior maxillary bone is the most common cause of orbital cellulitis, or orbital abscess, and reported two cases. Skillern claims that in infants there is no sinus of any importance, but that those that are present may be the starting point of an infection such as this, perhaps during delivery, or some infection soon after birth. He believes, however, that most of them are due to osteomyelitis rather than to sinus trouble. Posey stressed the point that dirty nipples or a breast infection of the mother may infect the child. In the case Dr. Yerger presented, after the baby developed its trouble the mother developed a breast abscess as a result of infection of her nipple from the pus from

the baby's mouth. Inasmuch as the sinus is not developed in infants the bone must be considered as being involved.

He said the last two cases represented his entire experience with osteomyelitis of the frontal bone and he had no mortality. There were two ways of looking at the problem from the standpoint of surgical treatment, one the conservative and the other the radical. He believed one should use a great deal of discretion and rely a great deal upon frequent roentgenograms to see whether there was any evidence of bone necrosis. The treatment which is generally advocated is very radical. Dr. Yerger did not think it should be treated so radically; in the beginning, where there is pus, the abscess should be incised and drained, just as an abscess elsewhere. When the infection is subperiosteal or extradural this must be taken care of, but he thought it a mistake to be too energetic in the removal of the doubtfully necrotic bone. Surgery should be limited to removal of the sequestra, and cases should be followed for from six months to a year by radiographic observation. If there was continued pus and the presence of sequestra, the condition should be cared for as it occurred.

**Radium in the Treatment of Carcinoma of the Tonsil and Larynx.**

DR. SAMUEL SALINGER presented a man, aged 58, engineer, who was first seen on January 22, 1925. He complained of hoarseness for over a year and of dyspnea for several weeks. Syphilis and tuberculosis were ruled out.

Examination showed a large, lobulated, irregular mass arising from the right side of the glottis, projecting upward above the level of the ventricular bands and almost completely covering both vocal cords so that only the posterior portion of the glottis was free. There apparently was no involvement of the arytenoid area.

A tracheotomy was done, following which a section was removed by suspension laryngoscopy and the growth implanted with eight radon seeds, totaling about four millicuries. During the next few weeks there was considerable reaction with some sloughing. The patient received three external radium applications over the larynx. On April 7th, under suspension laryngoscopy, the entire growth was removed and the base implanted with six radon seeds totaling three millicuries. Histologic examination of the mass showed a carci-

noma. Considerable sloughing followed and the patient was annoyed by a persistent cough. He received one more external radium treatment.

The slough gradually disappeared, and on September 7th a whitish mass was seen below the glottis, which was easily removed under local anesthesia, using the Haslinger, and proved to be a portion of the cricoid cartilage.

The patient had since gained 25 pounds and was in perfect health. His voice was hoarse but plainly audible. Examination showed that the greater part of both vocal cords were gone, and there was cicatrization below this point on the anterior wall of the trachea. There was the likelihood of a web formation at the point where the cricoid was lost and they were watching for this development.

Dr. Salinger's second case was a man, aged 59, night watchman, who was first seen on April 30, 1924. He complained of pain and swelling in the right side of the throat. Eighteen months previously he had had a fall, breaking off the stem of a clay pipe he was smoking, which injured the back of his throat. Three months later pain and swelling appeared. It was then diagnosed as syphilis and several treatments were given.

Examination April 30, 1924, showed a fungating, cauliflower, infiltrating mass, involving the right tonsil, the anterior pillar and base of the tongue. The right deep cervical glands were enlarged, dense and fixed. The Wassermann reaction was negative. Under local anesthesia, twenty radon seeds, totaling ten millicuries, were planted into the mass. The reaction which followed was very painful. The patient suffered a great deal in swallowing and lost considerable weight. The tongue was completely healed within ninety days, but the tonsillar area still presented some sloughing. At the end of six months this area also was healed, presenting a smooth, white scar, involving the whole tonsil region and extending through the anterior pillar, which was perforated up to the margin of the ascending ramus of the mandible. On account of discomfort resulting from food accumulating in this perforation the strand of pillar was later removed. The neck had been entirely free from adenopathy for nearly a year and the

throat presented the same appearance as it did when it first healed.

These cases illustrated what may be done with radium emanation. The principle of the treatment is to so distribute the agent within the growth as to thoroughly irradiate all parts of it with a minimum amount of damage to the neighboring healthy tissue. Failure results from the difficulty in accurately mapping out the extent of the lesion and from the general decline due to the dysphagia and other effects of the radium reaction. The problem is particularly difficult in the larynx because of the perichondritis which ensues, with the danger of necrosis of the cartilages. The best results thus far obtained in the larynx have been through the use of small intralaryngeal doses, supported by heavier irradiation from the outside.

#### **Intranasal Squamous Cell Carcinoma (Bilateral).**

DR. WALTER H. THEOBALD said that the case presented was of interest because of its rareness, its bilateral location, and especially because of its marked response to radium treatment.

A patient, male, aged 75, came under Dr. Theobald's care July 29, 1925, complaining of complete nasal obstruction and a mucopurulent discharge, which drooled continuously because of his inability to blow his nose. This had been present for a period of about four months and was first noticed after a severe attack of influenza, complicated with double otitis media lasting about two weeks. He stated that he had suffered from "nasal catarrh" prior to this trouble, for which he had never sought relief. He also complained of pain over the top of the head, inefficiency at his work and a loss in weight amounting to twenty pounds.

Upon inspection externally, the nose appeared swollen over the bridge, which was continuous with a puffiness below the eyes. Lacrimation was present, and his voice had the characteristic lack of resonance found in nasal stoppage.

Anterior rhinoscopy revealed a large papillomatous, pale red mass, completely filling both nostrils. This mass had the appearance of granulation tissue, intermingled with nasal polypi, and seemed to be intimately connected with the lateral wall of the nose as well as the septum. It was covered with a muco-

purulent secretion, was soft and spongy and bled rather freely when probed with an applicator. No ulceration was present. It was impossible to introduce the nasopharyngoscope on account of the bleeding, so that the origin and exact distribution of the tumor could not be determined. Cocain had no effect upon reducing its size. There were no enlarged cervical or maxillary glands palpable.

Laboratory examinations and the pathologic report were as follows: Urine and blood count negative. Wassermann test negative. The roentgen ray showed appreciable opacity in both maxillary sinuses, which was more pronounced in the right. The right frontal sinus also was somewhat opaque, and there was a question of certain ethmoid cells. The left frontal sinus seemed clear. The sphenoid sinus looked clear. There was nothing about the appearance of the sinuses that would argue toward malignancy.

On August 3, 1925, the patient entered St. Luke's Hospital for the purpose of making a histologic study of the tissue. Suitable specimens were removed from both sides of the nose under cocain anesthesia. The hemorrhage was brisk, and gauze packing in the nose was necessary for the following thirty-six hours.

The report of both specimens showed squamous cell carcinoma. The case was decided to be inoperable because the growth involved both nasal cavities and extended more or less into the ethmoid and maxillary sinuses. Also, the patient's weakened condition and his age contraindicated any radical procedure. Therefore, destruction of the growth by radium was considered the best means of treatment.

On August 11, 1925, radium was placed in each nostril for six hours, the dosage being 300 milligram hours. The radium tube was forced well into the growth on each side. Considerable reaction followed—swelling of the face, headache for several days and profound weakness.

On August 24th, 50 milligrams of radium was used in the right side and 25 milligrams in the left side for six hours. Dosage of 450 milligram hours. The reaction was not as severe and there was a marked diminution in the size of the growth.

On November 11th, 25 milligrams of radium were used in each side for six hours. The total dosage to date had been 1,050 milligram hours. Screening used was .5 silver and .8 rubber.

The condition of the patient when presented was one of great improvement. He attended his office daily, had gained fifteen pounds in weight, complained of no headache and breathed freely through his nose. Examination showed fibrous tissue and a firm, rather pale mucosa on both sides. There was plenty of room for breathing and an adhesion of the inferior turbinate to the septum on the right side.

#### DISCUSSION.

DR. NORVAL H. PIERCE recalled three cases that had come to his attention since the war. Two of them involved the ethmoid labyrinth, one the ethmoid on one side and the antrum. The first case was about of a man about 40, a bank employe, well nourished, who complained of stoppage of one side of his nose and discharge of mucopus, dull, heavy pain in the right side of his head and in the eye. Rhinoscopy disclosed a growth occupying the entire right nasal chamber down to the middle of the upper portion of the inferior turbinated body. This was polypoid tissue that seemed to be undergoing fibrous change, and between the lobules granulation tissue could be seen. He thought the process was an ethmoid polypoid degeneration. He removed the growth and did a complete exenteration of the anterior and posterior cells of the labyrinth. The tissue was sent to the laboratory and examined by competent men, who reported carcinoma. The patient had been under observation since then, and there had been no sign of recurrence in two or three years. No radium therapy was employed, and the operation was simply the ordinary intranasal removal of such a growth.

The second case occurred in a man in the seventies, who had a bulging antral wall, a slight displacement of the eye upwards and outwards, a quantity of very vile smelling pus and a mass occupying the entire left side of the nose. There was a suspicion of carcinoma. The growth was removed intranasally, the antrum opened and a great quantity of foul smelling material was discharged, which contained cheesy material.

The diagnosis from the pathologic laboratory was carcinoma. The patient was treated with three or four implantations of 50 milligrams of radium in the antrum but not in the nose. There had been no recurrence and the patient apparently was well.

The third case resembled the first. There was a diagnosis of carcinoma from the pathologic laboratory. The patient had received no radium therapy but had made an apparently permanent recovery.

Dr. Pierce thought two inferences could be drawn from these cases and that of Dr. Theobald: Inflammatory processes in the mucosa of the nasal accessory sinus may cause changes in the arrangement of epithelium which are similar to that found in carcinoma; or else the carcinomas in this region are exceedingly benign and, while carcinomatous morphologically, possess little or no malignancy.

Dr. JOSEPH C. BECK thought the point that there are histologic changes which are imitated by artefacts was very important, and this occurs not only in grouping but in section. The all-important point was that the diagnosis of carcinoma should include a statement of the type of cell. There are at least four clearly differentiated types, and the prickle celled, the basal celled, the squamous celled and the adenocarcinoma all respond differently to roentgen ray and radium. The change in the technic of treatment has been pronounced, and men who have studied many of these cases from the standpoint of dosage and technic have found that many mistakes were made in the early days of this treatment. The present day ampules are considered much more efficacious than the needle or external application of screened tubes. While there is danger from the cicatrization of the cartilage, Dr. Beck thought everyone would agree that this is better than having to do a laryngectomy, no matter how successful the operation may be.

Dr. GEORGE W. BOOT stated that Dr. Freer had treated two cases for him, with apparent cure, but both the patients had died later from recurrence. One patient who was treated by Dr. Simpson was still living, but in that case he believed there was a mistake in the diagnosis and that it was not a case of carcinoma.

DR. SALINGER (in closing) said that he had seen the patients referred to by Dr. Boot, who were treated by Dr. Freer and apparently cured, on several occasions. The only explanation was that the initial dose was so large that there was a subsequent breaking down of the tissues and the patients died of perichondritis and infection. One millicurie of radium emanation buried in the tissues will exhaust itself in thirty days, yielding a total of 133 millicurie hours. The principle of the treatment is to distribute the seeds in the infected tissue, figuring that the ampule has an active radius of about one centimeter in every direction.

DR. WALTER H. THEOBALD (in closing) stated that the pathologic report in his case came from Dr. Hirsch of St. Luke's Hospital, and the tumor had all the features of malignancy. According to LeCount, many such cases of carcinoma in the nose are amenable to radium treatment. No operating was done, a specimen being removed from each side of the nose with as little trauma as possible. Whether or not the growth was malignant, it had disappeared, and the patient had been able to breathe freely since the radium was used.

#### Parotid Fistula.

DR. GEORGE W. BOOT presented a patient who had been shot in the cheek and a parotid fistula followed. The area had been curetted, roentgen ray and radium therapy applied, without effect. He eventually took a heavy, braided silk thread and thrust it through the cheek, and left it in for thirteen weeks to establish a new duct. Upon removal of the thread the fistula closed and had remained so since. The patient was now under treatment for repair of the scar.

He had a similar case in a child following a mastoid operation done by another physician, with a fistula behind the ear, which persisted for a year.

#### DISCUSSION.

DR. CHARLES H. LONG recalled the case of a woman who was choked by her husband, causing traumatism of the left parotid gland. She had some paresis, great swelling and pain and along the lower angle of the inferior maxillary a fluctuating spot was found, which was opened. By keeping this open and



clean, the fistula healed in about two years, with little scarring or deformity. In his opinion, it was better to let the parotid gland get well by itself, if possible.

DR. GEORGE W. BOOT (in closing) said he did not present the case to demonstrate a new method of treatment, but because the treatment was old and still good.

#### **Suppurative Otitis Media.**

DR. NOAH SCHOOLMAN presented a patient with suppurative otitis media of fifteen years' duration, who came to him complaining of occasional vertigo. Examination of the ear revealed little except a small crust. Upon removal of the crust there was a little pus on the inner side and some glistening material toward the attic which, when removed, showed an opening into the attic, and the attic filled with cholesteatoma. This work made the patient dizzy, and on trying the turning test he found nystagmus with the slow movement to the opposite side. He considered the case rather unusual because of the problems involved in the treatment. All that could be seen was the perforation into the attic, but he was suspicious of sequestra in the labyrinth. The patient was in good health and attending to business in the intervals between attacks. He had no spontaneous nystagmus.

#### **Lung Abscess.**

DR. EDWIN MCGINNIS presented a series of lantern slides demonstrating lung abscess from various causes.

The first case presented was that of a boy of nine, with lung abscess caused by the inhalation of a piece of grass with which he had been picking his teeth. The condition reacted favorably to bronchoscopic aspiration and neosalvarsan.

The second case was that of a girl of six with a right lower lobe lung abscess following tonsillectomy. This was cured by two bronchoscopic aspirations and four injections of neosalvarsan.

The third case was one of right lower lobe abscess following tonsillectomy, but not caused by the anerobic bacteria. This reacted very well to two bronchoscopic aspirations.

The fourth case was that of a girl of six with three lung abscesses following tonsillectomy, one in left upper lobe, one

in right upper lobe, and one in the right lower lobe. These cleared up with neosalvarsan and bronchoscopic aspiration.

The last case was one of massive collapse of the left lung following the aspiration of a piece of peanut candy. Following one bronchoscopic aspiration the lung started to expand, and it was only necessary to aspirate once more to accomplish a complete filling out of the lung.

#### DISCUSSION.

DR. GEORGE W. BOOT objected to the nomenclature. In his opinion, unless the lung tissue is broken down it is not lung abscess but suppurative bronchitis, peribronchitis and pneumonia.

DR. LAURENCE H. MAYERS said that, in his opinion, it would be as easy to differentiate between a yell and a scream as between a bronchial pneumonia and lung abscess at such an early stage of the disease. If the roentgenograms Dr. McGinnis had shown were lung abscesses, he would have to explain more in detail how he had arrived at the diagnosis, as Dr. Mayers knew of no way, clinically or by the roentgen ray, that lung abscesses can be diagnosed at such an early stage of the disease.

DR. EDWIN MCGINNIS (in closing) agreed with Dr. Boot that in the early stages the cases are not lung abscess but inflammations of the mucous membrane. Anerobic infections, if permitted to go on, cause breaking down of the lung tissues, and the old picture of cavity with foul smelling sputum and all the other characteristic findings will be present. He believed that bronchoscopy would help these cases materially. A few years ago no one thought of using the bronchoscope or neosalvarsan in these cases, but the bronchoscopic aspiration has worked well in many cases, and if they can be treated early it will prevent the cavity formation. In a patient that had been operated on externally at the Presbyterian Hospital he found five or six bronchi spilling pus out into the main bronchus which the patient had to cough up. Early bronchoscopic aspiration would have helped this patient.

#### Osteoma of the Sphenoid.

DR. CARL H. CHRISTOPH presented photographs and related the case of a patient with osteoma of the sphenoid.

A woman, aged 45, had consulted him complaining of periodic headaches of greater or lesser intensity, sometimes accompanied by nausea and occasionally by dimness of vision, for the past eight years. She had experienced an especially severe attack during the past five days, accompanied by diplopia and very poor vision in the left eye. One brother had died of brain tumor, but aside from this the personal and family history was negative. Examination showed the left eye turned in, and the patient was unable to move it outward, but motion was normal in other directions. Vision in the right eye was 10/10, in the left eye 2/10, but the patient stated that she had always had good vision in this eye. Examination of the nose showed the septum deviated to the left, but no pus or other pathology was visible. The tonsils were small, submerged and contained no pus on pressure. Transillumination was negative. The color fields showed a contraction especially for form and somewhat less for green and red. Roentgen examination showed a bony growth in the region of the sphenoid sinus, evidently originating from the septum, separating the sinuses and apparently intimately connected with the lateral wall of the sinus. The blood and spinal fluid Wassermann reactions were negative. A diagnosis of osteoma was made.

Under local anesthesia the posterior one-third of the septum, with the rostrum of the sphenoid and the entire anterior wall of the sphenoid sinus was removed. Dental burs and forceps made no impression on the tumor mass, which was like ivory. A few chips were removed by means of a chisel, and the Lincoln-Gardiner Laboratory reported them bone tissue. The growth was so intimately connected with the lateral and superior walls of the sinus that it was decided to leave it in situ, with the hope that it would grow anteriorly. Three days after operation the external paralysis and the headache disappeared and the vision in the left eye was 8/10. Several subsequent examinations showed the color fields and vision to be normal and there had been no recurrence of the headaches.

#### DISCUSSION.

DR. OTTO STEIN considered this a very unusual case and said he had not before heard of osteoma confined to the sphenoid sinus, but had seen them in the nose and antrum and

frontal sinuses. He was curious to know how the diagnosis was established before operation. The photographs and the ophthalmoscopic examination suggested the findings in hypophyseal disease, particularly upon looking at the lateral view of the sella, where it encroached on the sphenoid sinus, but the operative findings proved the growth to be an osteoma.

DR. CARL H. CHRISTOPH (in closing) said the diagnosis was made chiefly by roentgen examination, which showed a definite tumor in the region of the sinus, and chips removed from this mass were reported to be bone. The only thing it could have been confused with was exostosis, and he did not know how one could differentiate those two conditions preoperatively.

#### Ragweed of the Chicago Area.

DR. ALFRED LEWY presented specimens of ragweed collected near his home, among them the most prolific producers of hay fever.

These three mounted specimens, the low ragweed (*ambrosia artemisiifolia*), the giant ragweed (*ambrosia trifida*) and the burweed marsh elder (*iva xanthifolia*), represented the principal offenders in this region as the supposed cause of the autumn type of hay fever. They are common on all empty lots, along ditches, etc., and their pollen, which is very profuse and light, is wind borne to enormous distances and great altitudes.

#### DISCUSSION.

DR. EDWIN MCGINNIS was much interested in the presentation, for he has been investigating ragweed for some time. He agreed that such weeds are the predominant causes of hay fever.

DR. H. L. HUBER thought that another one of the ragweed family should be included in this collection, the common cockle bur. It is widely enough spread to be of importance in some cases. Another ragweed, the *ambrosia psilostachya*, or western ragweed, is found in a few places. Dr. Lewy had included one of the newer ragweeds, the *iva xanthifolia*, which has become very important in the Chicago territory recently, as it is invading Chicago from all sides and spreads very rapidly.

DR. ALFRED LEWY (in closing) said that of course the cockle bur (*xanthium canadense*) is a closely related genus, but he

did not think its pollen was of as great importance as the three mentioned. He was not aware that the western ragweed occurred in the Chicago area, as mentioned by Dr. Huber. This presentation was not intended to cover all of the pollen bearers concerned in autumn hay fever, but was merely for the benefit of those members who do not get out into the great outdoors much and who might be interested in getting acquainted with the source of much trouble to some of their patients and to themselves.

**Cervical Emphysema Following Tonsillectomy.**

DR. AUSTIN A. HAYDEN presented a patient who had recently had a tonsillectomy under general anesthesia. The patient was one of the resident staff at St. Joseph's Hospital, and the evening after the operation Dr. Hayden received a telephone message from one of the interns, stating that the patient had some air in his neck. Upon going to the hospital he found the neck somewhat swollen, and the patient had discovered crepitation on feeling his unshaven face. Dr. Hayden found the crepitation very distinct, questioned the patient carefully as to whether he had been coughing and was told that he had coughed a great deal in an effort to raise some dry mucus that had interfered with breathing. There was very little swelling in the throat and the mucus had been expelled. By means of the application of ice and thoroughly expectant and confidence inspiring treatment the patient made an uneventful recovery.

Dr. Hayden thought that the question of air in the tissues following nose and throat operations was somewhat interesting. He cited the case of a clergyman who came to him a number of years ago to have his eustachian tubes inflated. He had done this a number of times, but one day when he introduced the catheter the patient exclaimed, "Doctor, I am choking to death." His voice was perceptibly thickened, and upon taking a tongue depressor and examining the back of his mouth Dr. Hayden was amazed to see that the uvula was tremendously edematous and that there was a large amount of air underneath the superficial cervical fascia. Evidently the catheter had penetrated the membrane around the mouth of the tube and some air had been forced out, which rapidly spread all

over his neck. That patient also made an uneventful recovery, but probably if any infectious material had been blown out a Ludwig's angina would have followed.

The essential thing is to see that tonsillectomy patients do not cough violently or clear their throats following operation.

#### DISCUSSION.

DR. ROBERT H. GOOD said he had had the same thing happen in the case of a woman under local anesthesia, with the same symptoms, and an uneventful recovery.

DR. HARRY L. POLLOCK said one of the most extensive cases that he had seen followed a tracheotomy, when the tissue was closed too tight around the tracheotomy tube. Within an hour the entire neck was full of air, but the only treatment necessary was to open one of the stitches. In another case a child had inhaled some small pieces of cork, and the case was diagnosed as pneumonia, but emphysema was found above the clavicle. This was probably due to the forced breathing, for air could get in but none could get out. After the bronchus was opened and air, by suction of thick mucus entered, the condition subsided very rapidly.

DR. AUSTIN A. HAYDEN (in closing) cited another case seen a number of years ago. A lady called him by telephone and said that "her husband had blown his eye out when he was blowing his nose." The patient was the driver of a milk wagon, and, in unhitching his horse, he had received quite a severe blow on the nose as the horse turned his head to get rid of a fly. During the night he blew his nose quite forcibly, and evidently some air entered the tissues and filled the entire area about the eye, because of a rupture of the mucous membrane.







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